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THE EARLY RECOGNITION OF OTITIC INTRACRANIAL COMPLICATIONS.*

DR. V. V. WOOD, St. Louis.

To make a diagnosis when any disease is still in its infancy or before the full development of its characteristic clinical picture is often difficult, but always extremely desirable. The importance of an early diagnosis is enhanced considerably in a serious malady. It becomes a protection to the physician against adverse criticism, regardless of the outcome of the illness. The result, if bad, then assumes more of an inevitable aspect as all parties concerned should realize that no time was lost in adopting the proper line of treatment.

I can think of no other condition in which an extremely early and accurate diagnosis may be of greater importance than in otitic meningitis. Some brain surgeons maintain that the best chance for successful surgical intervention is lost if they do not get the opportunity to operate on such a patient during the first few hours after onset of the disease. Eagleton and others propose to attack the infection at its portal of entry to the meninges before the early localized process has had time to spread to a general one. Such a course, naturally,

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depends primarily upon an early correct diagnosis. This diagnosis must include not only recognition of the type of lesion, but also an accurate opinion as to the location of the portal of entry.

A brain abscess patient, however, may do well even when the diagnosis is made rather late because it usually takes considerable time for an abscess to form, to localize, and to reach a stage when surgical intervention will be successful. Such a patient usually does better if operation is deferred as long after the diagnosis is made as his condition will permit.

The management of any case is more intelligent after a correct diagnosis has been made. Even though a definite opinion cannot be formed at first as to the exact nature of the impending intracranial complication, it is a great help to recognize, at the earliest possible moment, that something of the sort is on the way. The physician is put on guard and he begins to tune up his diagnostic ability to the highest pitch. He should then see the patient oftener and examine him more fully. I fairly live with some of these patients, frequently finding it necessary to visit them three, four or more times daily. In that way many signs, which are more or less transient at first, are caught. Symptoms may be found that others cannot recognize and if the suspected complication turns out to be meningitis, a continuous spinal drainage, a continuous cistern drainage, or some other operation aimed at subdural drainage, may be advised at the earliest possible moment.

It should be kept in mind that this paper is concerned chiefly with the early recognition that some intracranial complication is on its way, rather than the appearance of the fully developed clinical picture. Therefore, many pathognomonic symptoms and signs appearing later will not be emphasized. They are fairly well agreed upon and familiar to all.

Neither is any attempt being made to review the literature and to set forth the latest ideas of other men. Some observations, perhaps considered of great importance by others, will not be mentioned because they did not appear or, if so, escaped notice in my series of cases.

Further, the points discussed are chiefly those to be picked up at the bedside without the aid of elaborate laboratory apparatus or complete hospital equipment.

A diagnosis of the exact type of intracranial lesion forming may be impossible during the early stages. Even when there is ample justification of a firm conviction that an intracranial complication is impending, it may be absolutely impossible to determine for several

days just what kind of lesion it will be. The onset may not be typical of the ultimate development and again, even though the correct final diagnosis is clear from the outset, there may be no early clue to the portal of entry. It is especially difficult to visualize the route of invasion and the complete clinical picture prior to operation. I find it much easier to "hunch" (I can think of no word as good, slang though it may be) the entire syndrome correctly after an operation than before. More will be said about that later, but observations made during the operation may help immeasurably in estimating the correct portal of entry of the intracranial infection.

Some cases are branded with definite and some with rather vague indications of their unfavorable course, almost from the beginning. Others apparently do splendidly, with no indications whatever that all is not well until days, weeks, or even months, after an uneventful operation and early postoperative course. Even in one of these cases a diagnosis can be made very late in the course of the disease and still be considered early, or at least timely, if it comes to the physician a considerable time prior to the appearance of the fully developed clinical picture of the terminal complication. Therefore, an early diagnostic hunch may force itself upon one at the very first observation of the patient or at any subsequent visit. At times it will hardly bear close scrutiny and analysis. It may not sustain itself with definite reasons that can be set forth intelligently; *i. e.*, sometimes one is puzzled to explain the wherefore of it either to himself or others, but it may remain and be correct. Perhaps, at times, the knowledge that the existing epidemic is of a virulent type and that a large percentage of cases are doing badly may help a physician to sense subconsciously that a certain patient is too ill to fight back well, or, in some rather indefinite way, he may resemble another patient who went wrong. Severe intracranial complications usually run in epidemics. Sporadic cases are always with us, but the general type of case in each seasonal epidemic is very constant. During certain years there are many suppurating ears, but few mastoids. In other years, perhaps, mastoids are numerous, but of a mild type and few intracranial complications occur. Then a more severe epidemic appears, due to more virulent organisms, and a large percentage of deaths from intracranial complications is seen.

The subjective symptoms may furnish the only early clue to the extreme seriousness of the case. Upon examination of the patient absolutely nothing may be found to explain the symptoms complained of by him. If he is seen early, at the very onset of his illness, a record of these complaints, kept in chronological order as they appear,

may be of inestimable value when reviewed in the light of later developments. If the patient is not seen until later in the course of his illness, the initial history obtained from him or some other dependable party is the only means of knowing many things that may have gone before. Therefore, the initial history may be an important document; but, on the other hand, it may be misleading. Its importance bears a direct relationship to its accuracy, but it should always be taken carefully and should be gone over and checked repeatedly at later dates.

The real worthwhile data may be hard to obtain and sometimes it is more difficult to eliminate the confusing and misleading features of a history than to pick out the helpful ones. Many important symptoms and signs may pass unnoticed by untrained observers and much emphasis be placed upon unimportant and trivial matters. When the patient is an infant or young child, too much dependence cannot be placed in what the parents or the nurse have to say. Sometimes it is even difficult for them to tell that the child has ear trouble until the ear begins to discharge. They may note that he pulls at one or both ears and is in pain, but many important signs are often overlooked, even by intelligent people. For instance, I once saw an infant dying of meningitis who undoubtedly had begun projectile vomiting forty-eight hours before. He probably had a stiff neck and a spasticity of one entire half of the body for twenty-four hours, and still the seriousness of his condition was not recognized until a convulsion occurred and he passed into coma. Then he was brought to the hospital.

Accurate data on the number of previous ear infections, their severity, and the exact date of each, may be difficult to obtain. This information is usually of great importance from a diagnostic or prognostic standpoint. The entire aspect of the situation in a given case may hinge upon the question of whether or not the attack under observation is a primary one. Not only may the diagnosis and prognosis depend upon that question, but also the method of handling. The exact date of the last ear infection may be extremely important if it has been recent. With the same clinical picture presenting in both instances, early operative interference is much more justifiable in a second, third, or subsequent mastoid infection than in an initial or primary one and an operation may be urgently indicated if the last one has been recent. Justification for early operation increases with each recurrence. Even though prior infection of the mastoid cannot be definitely established, any former attack of otitis media (whether suppurative or nonsuppurative, but especially in the for-

mer) may have been accompanied by considerable unrecognizable mastoiditis. The healing process may become arrested at any stage short of complete repair and remain as a partially walled off, dormant area for years. It may be situated very close to the dura and its dural aspect may be very weak or deficient and offer the avenue of least resistance for a break. Such an area can be quickly lighted up by a new infection and resume its progress where the old ceased.

When there are several children in a family the parents often become confused as to which of them have had previous ear trouble. The children, themselves, may not remember. They may have been too young when affected. For example, in two cases of mastoiditis, one of which returned a pure culture of diphtheria bacillus and the other a pure culture of colon bacillus from the mastoid at operation, it was impossible to determine definitely whether or not there had ever been previous ear infections. In each instance it was first denied, then it was admitted as possible, and finally the decision left in doubt because the family could not decide which of the children had been so affected. From a scientific view this point was very important in each instance because the bone destruction was almost unbelievable for the short time that had elapsed between the onset of the last earache and the operation.

It is not unusual for patients to become confused as to which ear had been infected previously. That is easy for anyone to do and the otologist may or may not be able to settle the question by his otoscopic examination.

It is frequently impossible to determine the exact duration of the present illness because the date of onset of earache or ear discharge may be missed several days. This is another very important piece of information because the duration of time between the onset of earache and the onset of mastoiditis or other complications of otitis media is frequently an index to the seriousness of the case. The earlier clinical signs of mastoiditis or other complications appear after the onset of tympanic infection, the more virulent is likely to be the organisms, regardless of whether or not there have been previous ear inflammations. It may be as serious in one instance as the other, but I believe they should be handled differently. The increasing urgency of early operation with each subsequent attack of mastoiditis has been emphasized strongly by Kopetsky and others. If an attack of otitis media with mastoiditis has followed closely upon another, I again repeat that an early operation may be extremely urgent or even life saving, but I believe the procedure will be useless or even harmful if the middle ear and mastoid never before

have been diseased. This is the time to wait for localization and if it does not occur the patient will die in spite of a mastoidectomy.

While many valuable moments are wasted listening to the useless conversation of patients, their relatives or friends, it is always well to try to find a few grains of wheat among the chaff. Some very important fact may slip out at any moment. Only a sentence or two may lead directly to the crux of the situation and guide one right or prevent one from going too far wrong. For instance, at three o'clock one morning I was called by another physician to see a gentleman, age 64 years, with a very definite mastoiditis and complaining of a terrific headache, which had persisted for days. He had a slow pulse (in the fifties and at times between thirty and forty) with a subnormal temperature. I suspected a brain abscess and so informed his physician. He was scheduled for operation later that day. His son met me at the hospital prior to the operation and said, "Now, doctor, don't let that slow pulse influence you unduly, because father has always had that. Every doctor who ever examined him has remarked about it." So it turned out. After a simple mastoidectomy his headache disappeared, all other symptoms cleared up promptly, except the slow pulse, which he still has. Within a week I was called by another physician to see a boy with a very evident mastoiditis, a temperature of 105°, and a history of having had several convulsions that day. He had been unconscious most of the previous twelve hours. Upon examination he presented a marked spontaneous nystagmus. His neck was very rigid. His entire body, arms and legs were markedly spastic. He could be aroused only into an antagonistic attitude and resisted all disturbing efforts at examination. When the lids were raised to examine the eyes more carefully, he shook his head and entire body and resisted everything. Needless to say, little time was wasted in trying to determine the character of the nystagmus. It was there, with many other things, and I immediately began to visualize an advancing meningitis with exudate probably creeping over the cortex, etc. The mother then volunteered the information that his eye twitchings and general spasticity had been present since 1918, at which time he had had an attack of infantile paralysis. After a simple mastoidectomy he became as well as usual, but walked with a stiff, spastic gait, had a distinct tremor and nystagmus, mask-like face and drooling at the mouth, but he was no different from what he had been for eleven years. He presented the picture of a postencephalitic and I think his so-called infantile paralysis was undoubtedly an encephalitis. The convulsions probably were due to his high temperature. The mother's remarks certainly improved the prognosis and quickly undermined my snap diagnosis.

Subjective Symptoms: Certain subjective symptoms of much importance may be submitted by the patient at the initial visit to his beside or during later contacts. It is always disturbing for a patient to complain of queer subjective sensations of touch, sight or hearing. Sometimes he notices that he does not see correctly, but may not be able to explain the exact nature of the difficulty. One of my brain abscess patients seemed to see normally when both he and the object were fixed so he could gaze at it steadfastly, but I do not think he picked up detail quickly when trying to focus on a new or moving object. He could read signs across the street from my office as well as I, but he maintained that something was wrong with his vision, although he could never clearly define just what it was. The oculist may find nothing objective at the first examination, or he may find a mild blurring of a disc, engorgement of vessels, or a slight weakness of an ocular muscle. The fields may be found abnormal or they may not. Photophobia may mean nothing as it accompanies even low fever in some persons and, again, it may be very significant. It nearly always precedes or accompanies intracranial conditions. It is frequently the first complaint and should never be passed over lightly. Anorexia, nausea or unaccountable vomiting are always disturbing. The so-called projectile vomiting is not necessary in early cases. Nausea preceding the vomiting has been characteristic in my early cases. They usually have turned pale, "sweaty" and then vomited. The nausea may be short and the vomiting may occur very quickly. The patient may deny or admit subjective nausea, but he "looks it," except in very advanced and typical cases. Only slight vertigo may indicate that the labyrinth is becoming involved or that a brain abscess or meningitis has already begun. Of course, some deafness usually accompanies a suppurating ear and means little unless it is excessive and is backed up by other signs of labryinthine involvement. Vague cephalic pains not amounting to actual headache are hard to interpret as of importance, but it is not conducive to ease of mind if they are too far removed from the ear.

Careful inquiry should be made as to chilly sensations or any intervals of coldness when the patient needed extra covers or heat applied, especially if others about the room were comfortable. A hard shaking chill will be mentioned usually, but the early chills are often slight and go unnoticed. They may indicate the beginning of a sinus thrombosis or any leak of bacteria into the blood stream. They may accompany any sudden extension of the infection. In children a pyelitis should be watched for very carefully and excluded before undue emphasis is attached to a chill. Central pneumonia

and other general septic complications should be thought of and looked for by a good internist, but the more negative the findings, the more significant the chill.

Convulsions usually speak for themselves and bring the doctor on the scene immediately.

Mental Attitude: Otitic infections, intracranially bound, usually leave their imprint on the patient's mental attitude as manifested by irritability and sometimes a rather vague apprehensiveness. Children often cling to their parents in fear and trembling or are morose, listless and disinterested in play. This may be most striking if the child has been known previously and has been seen through other rather severe illnesses when he was amenable to handling, cheerful and not distrustful of others. They cannot adequately explain their depression. That is most important. There is no apparent reason for the child to be depressed. This condition usually is very profound and nothing will lift the patient from the depths and the feeling of despair for any great length of time. Younger children cease to play or to react to outside impressions and diversions that usually interest them or bring them pleasure and ease of mind. Older individuals frankly discuss their thoughts and feelings, or speak of their belief that all is not well and that something is happening that will end disastrously. One patient of mine morosely arranged for the disposal of her best linens and other effects, in spite of the fact that for nearly two weeks her temperature was above 99°+ but once her wound was healing splendidly, and all apparently was going nicely, except that she complained of a rather persistent, unexplained headache. She died of meningitis after the third postoperative week. It is a queer fact that parents frequently overlook this change in mental attitude. They may be the last to admit it even when it is quite evident to others. A history from intimates that the child's mental attitude is normal must not be taken too seriously by the physician if he is differently impressed, even though he may not have known the patient previously.

Headache: General headache or localized cephalic pain far removed from the ear is not a good omen. After a thorough paracentesis or a free spontaneous rupture of the drum, patients with suppurative otitis media are usually fairly comfortable, except for pain well localized in the ear or mastoid from drainage block. Referred pain due to this development may be difficult to exclude if the cephalic pain is occipital or cervical or if there is a neuralgic type along the fifth, but wherever located, it should not be ignored if it extends too far from the ear. If the headache is temporal, frontal

or behind the eyes and is accompanied or preceded by severe photophobia, it is especially significant. A history that the patient is subject to headaches of similar character may relieve one's mind somewhat, but it should not be depended upon in the presence of a suppurating ear. Headache or severe pain radiating from the ear or mastoid in the presence of a profuse aural drainage is not good. Neither is it a welcome observation when scant drainage follows a thorough paracentesis. In either case, a mastoid block may be suggested and the blocked cell may be situated deeply and break the wrong way. The behavior of the headache is very significant. If due to intracranial infection it usually is constant and present at all hours of the day or night. Before taking on a typically cerebral character, it is difficult to relieve and comes back soon after the effect of any drug wears off. Occasionally it is less constant and more wavelike or intermittent.

Examination of the Patient: Many findings on examination would not be of great importance in themselves if standing alone, but when found present with others (perhaps just as insignificant in themselves) a syndrome appears. Therefore, it may be necessary occasionally to wander off the subject at hand of merely describing a finding, in order to fill in missing parts of the complete picture. Many trivial things become significant when properly connected.

General Appearance of the Patient: The general appearance of the patient frequently is suggestive. It is a good plan before beginning the real examination (especially if the history has been obtained from someone else and the physician is entering the sick room for the first time) to stop by the bedside and engage the patient in light conversation, if he will, and look him over casually, but carefully, from a slight distance. When going bad these patients usually lose color and vitality rapidly and appreciably. Even when the color and weight is not noticeably affected, many of them appear unusually prostrated. Occasionally one of them may go almost to the final day without much visible failing and it is hard to realize that he is approaching death rapidly, but such a patient usually does not linger long and is carried off quickly.

Cranial Nerve Disturbances and Ocular Palsies: The patient should be examined carefully and frequently for cranial nerve involvement and weakness of ocular muscles. A slight weakness of the seventh, caught early, may be most significant. While involvement of the sixth has little or no localizing value, it is intensely disturbing to find it even slightly affected in a suppurating ear condition. It was absolutely the first definite warning signal in one of

my patients who eventually died of meningitis. The size, equality and reaction of the pupils should be watched carefully each day.

The neck spasticity and Kernig's sign should be tested for meticulously and checked frequently. The first minute onset of spasticity may be caught instantly because a slight change has been noted over the previous examination. An excellent consulting neurological surgeon may be unable to verify this observation at his initial examination, whereas the man in constant attendance is fairly sure of it.

Pulse: The pulse cannot be watched too carefully or taken too often. It always should be taken steadily for one or two minutes (not for ten or fifteen seconds and then computed). After the patient has been hospitalized or placed under careful and competent nursing, study of the chart may reveal an early relative slowing of the pulse curve with or without moments of actual slowness. There may be either a definite dropping of the pulse rate over what it has been or it may be relatively slower than it should be when compared with the temperature curve. Taking the pulse continuously for one or two minutes and frequently repeating the procedure may catch moments when it shows a tendency to slow very perceptibly or be actually slow. These changes may be traced on a well kept chart for days before a constantly slow, typical cerebral pulse appears.

Furthermore, I now always check an exceedingly slow pulse with a stethoscope over the heart. July 13, 1930, I sent a patient to St. Luke's Hospital for observation, after making a tentative diagnosis of some beginning intracranial complication because twice I caught the radial pulse rate below sixty. There was no mastoid tenderness, no otoscopic evidence of mastoiditis (except a rather profuse aural discharge), but the patient complained of a rather persistent headache starting in the frontal region above the left eye, radiating backward over the ear and downward into the neck on the left side. I had noted no pauses or intermittency in the pulse as one should if extrasystoles were present, but when examined carefully in the hospital by Dr. A. B. Day such a condition was discovered. Many heart beats were not reaching the wrist. The patient died of meningitis on July 28, 1930, and it was never known whether my observation of a slow pulse on July 12 was accurate or not. It proved a good hunch, but may have been founded on a mistake.

While this relative slowing of the pulse curve is very significant when definitely present, it is well to issue the warning that occasionally a meningitis case appears, runs its fatal course, and comes to a postmortem, which proves the diagnosis, without ever exhibiting a really slow pulse. This only emphasizes the trickiness of intra-

cranial lesions. No one supposedly pathognomonic finding is dependable.

There also may be detected a bound, or sharp cerebral feel, to the pulse quite a while before it actually slows and becomes typical. A kidney pulse is similar in character, but usually feels harder and tighter during diastole. If there is no chronic hypertension evident and the urine is normal, it is disquieting to detect an apparent change to a cerebral bound in the pulse.

The Temperature Curve: The temperature curve may be misleading prior to the definite onset of intracranial complications and even thereafter. The typical subnormal temperature of a well localized brain abscess needs no comment. A high temperature is not pathognomonic of serious complications setting in, nor is a low, partially subnormal curve an assurance of safety from meningitis. The temperature may fluctuate for days, and the peak of no daily curve top 99°, and yet the suspected brain abscess suddenly may flare up into a rapidly fatal meningitis and the autopsy may show definitely that a partially localized meningitis had been smoldering for a long time previously. Pneumococcus Type III infections especially are prone to be sneaking and silent (I can think of no more appropriate word than sneaking) and a fully developed meningitis may burst forth suddenly in a patient who apparently had been doing splendidly. Prodromes and warning signals in such cases are difficult to detect. A sudden, inadequately explained temperature rise of even two or three degrees is always a cause for concern, either prior to operation or after. The less apparent reason for it, the more significant it should be considered. It may mean any sudden extension or breaking through of the infection to a new area.

Respiration: Fully developed intracranial complications often produce Cheyne Stokes breathing and respiratory irregularities, but I do not recall having seen any significant early changes.

It is not necessary to devote much space to a discussion of the appearance of the drum membrane and external canal, the mastoid examination findings, character of the aural discharge or the bacteriology of the middle ear and mastoid secretions. That goes back to the fundamentals of otology in which these findings are a matter of common knowledge and their positive significance well agreed upon. Generalizing a bit, it may be stated that when any well recognized sign or symptom of extension of the infection appears soon after the onset of earache or is rapidly progressive when appearing later, it should not be ignored, especially if accompanied by other suggestive phenomena. Only a few "pet observations" will be mentioned.

Otological Examination: In an evidently sick and septic child, if the drum has lost its red inflammatory color, presents a dry, dull yellow, lustreless or "fuzzy" buckskin appearance, the hearing is down and after paracentesis the exudate appears coagulated or does not follow the knife readily, further complications should be watched for carefully. I have found that picture more than once with an insidious Pneumococcus Type III infection.

The so-called myringitis bullosa picture, with dark hemorrhagic blebs on the drum, may be indicative of a virulent infection. They usually occur in influenza patients, but early cultures nearly always bring in a streptococcus. I do not incise the drum in such cases until the hearing declines. The hearing may be up the first few hours and fall rapidly thereafter. When the hearing decreases markedly a middle ear infection nearly always supervenes (although there may be some diminution of hearing, due to loading of the drum, in any myringitis bullosa, especially if the blebs are large). Mild cases of myringitis bullosa run very little temperature. A high temperature when the hearing is still good usually means that the infection is more virulent than the average (assuming, of course, that there is no other complication present, such as a follicular tonsillitis, to explain it).

Regardless of the amount of drainage from the tympanum, if the well known beefy thickening of the drum, especially in the upper quadrants, appears early or progresses rapidly after a late appearance and repeated paracenteses fail to bring about its collapse or establish the bulging appearance as due to pocketing, it may be the first indication of rapid extension of the infection. There may be no positive mastoid tenderness or other signs accompanying the occurrence of this condition, but it is of double importance when accompanied by other disquieting developments. When occurring late in the disease and progressing gradually, it is of no significance as an indication of virulency, because it is a common and constant finding in cases developing a mastoiditis.

Mastoid Examination: If mastoid tenderness is localized chiefly over the emissary vein, it signifies that infection is near a wide open gateway to the intracranial structures. It may be the first and only early forerunner of serious complications.

Lack of mastoid tenderness should not mislead the physician if there is deep-seated subjective pain in that region or if the picture presenting through the ear speculum (beefy drum, sagging posterior superior canal wall, excessive aural discharge, etc.) definitely indicates an early or rapidly progressing mastoiditis. Especially is this

a bad omen if the patient is morose and complains of photophobia or a constant general headache at a time when the average patient should be fairly comfortable. As a general rule, I would much prefer to find exquisite mastoid tenderness than none at all if other indications of mastoiditis are present. Lack of mastoid tenderness may be found when there is considerable destructive mastoiditis (often penetrating to the dura as an extradural abscess) and be explained at operation by finding a very thick and firm cortex, which masked the underlying destruction. Mastoiditis is occasionally very patchy and often for no apparent reason. Sometimes, when the cortex is not especially thick, there is no external tenderness because all the infected cells are placed deeply. Not only may the infection begin deeply, but it can remain so and the superficial cells escape altogether. Mastoid tenderness may not be elicited until the cortex and periosteum are approached by the infection.

Characteristics of the Aural Discharge: The significance of an excessive amount of aural discharge (more than possibly could be produced in the middle ear alone) is well known. It is also generally accepted that the character of the discharge may indicate definitely the predominating type of organism, *i. e.*, streptococcus, pneumococcus, staphylococcus, etc. The appearance of the aural discharge certainly should be noted carefully and duly respected, but it may be misleading from a prognostic standpoint. Just because a streptococcus is indicated, it does not always mean that particular strain is virulent. The pulse, temperature and other clinical signs or symptoms should all coincide to complete the picture. A constantly hemorrhagic discharge, with few leukocytes at any time, may be accompanied by an entirely different type of mastoiditis. Especially may this be true when there have been previous mastoid infections. Various missing parts or confusing angles to the general clinical picture may be so explained at operation. I had one very striking case of this sort, but the mildness of its course (low temperature, lack of prostration, etc.) did not fit in with the virulent appearance of the discharge. The innocent appearing, light gray mucoid discharge so often seen with Pneumococcus Type III infections is always a cause for uneasiness and the absence of all other signs of virulence should not be considered as an assurance of safety until weeks after the patient apparently is well.

Turning, Caloric and Galvanic Reactions: Volumes have been written on the turning, caloric and galvanic reactions. They are of great localizing value in intracranial lesions. Their importance in making early diagnoses may be considerable, but is chiefly confined

to selected cases. They should be done and interpreted only by men familiar with them. Of course, turning is not usually practical in an acutely ill, bedfast patient, but distinctly indicated caloric tests can be done usually at any time, regardless of whether the patient is ambulatory or not.

Nystagmus: I have never seen spontaneous nystagmus as the initial symptom in an acute intracranial case unless it accompanied vertigo or vomiting. It has always been preceded by something else and usually an intracranial complication was suspected before spontaneous nystagmus put in its appearance. It may be an initial symptom of labyrinthitis, but does not necessarily foretell that an intracranial extension will occur.

Aphasia: Much the same can be said of aphasia as was just previously stated of nystagmus. Even in left temporal lobe lesions the aphasia has always been preceded by some other suggestive symptom (perhaps only photophobia, slight temporal or frontal headache, change in mental attitude, etc.).

Nystagmus or aphasia may have actually appeared first in some case seen later when it was impossible to determine accurately the sequence of events from the history, but never has either occurred first in one that I have been able to watch from the onset. Others may disagree with that statement as they may have had different experiences.

Bacteriology of the Secretions from the Middle Ear and Mastoid: To be reliable, all slides and cultures of the middle ear secretions should be taken as soon as possible after the discharge begins. Whenever possible, they should be made at the initial paracentesis, and if not then, as soon afterwards as can be arranged. If the patient comes in late, after the ear has been discharging several days, the bacteriologic findings are not dependable because much secondary infection may have set in. This secondary infection may quickly overshadow or blot out the primary organism.

Cultures should be grown on several different media. The predominating organism may be missed if only one type of culture medium is used. Dr. James Costen, of St. Louis, reports a case of diphtheritic mastoiditis in which the causative bacilli were missed at the first culture because only one type of medium was used. No comment is necessary about the significance of finding a Pneumococcus Type III, or virulent form of streptococcus, in either the aural or mastoid cultures. It should be emphasized again that these two cultures (aural and mastoid) may return different organisms and thus explain confusing angles to the previous clinical course. The

silent treachery of the pneumococcus has been stressed, but it should also be mentioned that streptococci are not always open and stormy, but may ape the former.

Lumbar Puncture: Early lumbar puncture has been criticized and advised against on the theory that such a procedure might spread a localized meningitis, disturb or excite a small area of encephalitis, or remove pressure from a localized brain abscess. It is not a good plan to unnecessarily traumatize or stir up the central nervous system, but formerly I was much more cautious about doing early lumbar punctures than I am today. I still prefer not to do them if brain abscess is suspected, but I now do lumbar punctures without hesitation at any time in suspected meningitis, if it appears to be the only means of getting early information that can be used to advantage in determining how best to handle the case. They should never be done promiscuously and if they do not promise to give that kind of information it is better to wait indefinitely. At times an early lumbar puncture is the only means of which I know of definitely determining at once whether a spinal drainage, cistern drainage, or some other surgical procedure is imperative, unnecessary or useless. Without going into detail as to the chemic, pathologic or bacteriologic findings, the chief significance of a lumbar puncture is usually in whether or not bacteria are present in the spinal fluid. In that respect, a spinal puncture may offer a great aid in prognosis. No matter how many cells are found, what their character may be, or what the chemistry may show, I never prepare to write a death certificate on a lumbar puncture finding until bacteria are found and identified.

Blood Examination: The blood picture may be very difficult to interpret. It usually is telling some story accurately and after the patient comes to operation or autopsy the entire previous behavior of the blood picture may be perfectly clear, but it is then too late to help in the early diagnosis or prognosis of that case. Much work has been done recently along that line and, while many papers are still somewhat contradictory, it does appear that at last we are making progress. It is probable that we are reaching a point now where the work is better standardized and interpretation of the different blood pictures soon will be well agreed upon by the various men. The older white blood counts and also the differential counts may be very tricky in their behavior. Of course, a rapidly rising white count to 30,000, 40,000 or above is a distinct danger signal and often just precedes other definite signs of meningitis, but a low count cannot be depended upon as a reliable indication of safety

when other clinical signs and symptoms do not coincide with it. One of my lateral sinus thrombosis cases had three chills, followed by a rapid temperature rise to 106°+ each time, all within thirty-six hours, and still the white blood count dropped steadily from above 17,000 after the first chill to about 8,000 following the third. Her temperature dropped to normal forty-eight hours after opening the sinus and tying the homolateral jugular and it remained there. I have not yet had enough experience with the new Schilling hemogram, observations of the degenerative changes in white cells, etc., to discuss their value.

X-ray Findings: Accurate interpretation of X-ray findings depends upon so many factors that the data obtained may be priceless or misleading. A single set of plates may be positively confusing unless previous attacks of mastoiditis can be ruled out definitely (which is often impossible or extremely difficult). It is not always easy to differentiate between old and new pathology and passive processes may be misinterpreted as active ones. Only a series of plates taken at several day intervals are reliable. Even when there is every reason to believe that the attack of mastoiditis under observation is a primary or initial affair, the pictures may be days behind the actual pathological development. It is remarkable how far advanced a mastoiditis can be without much showing on the plates. In other cases the pathologic process is apparently exaggerated by the X-ray. It all depends upon the kind of pathologic condition produced. When clinical signs and symptoms disagree seriously with the X-ray findings, I am inclined to discount the latter insofar as it can be done with any degree of safety.

This is not an attempt to belittle the importance of X-ray pictures. Far from it. They should always be taken, studied carefully, and never should be thrown aside carelessly. Astounding revelations may come to light by X-ray when no other method of examination would have exposed them. The accurate location of the most advanced destruction may be helpful and point the way to what is to follow. Great destruction directly over the lateral sinus or an unsuspected removal of the inner table down to the dura may be revealed. When previous attacks of mastoiditis can be established definitely one is on guard in reading the pictures, but even when there is no history of previous mastoiditis it should be remembered that if any attack of otitis media has occurred a certain degree of accompanying mastoiditis cannot be excluded unless a series of plates, taken at that time or since, are available. No one can estimate how much clinically silent mastoiditis may have accompanied any previous ear inflamma-

tion. It always should be suspected if the preceding otitis media were suppurative and considerable suppurative mastoiditis may accompany or follow a nonsuppurative otitis media (although it is much more rare). The former mastoiditis may get entirely well or its resolution may stop at any stage short of complete repair and remain so as a dormant, smoldering process for years. These old areas may be misinterpreted as recent destruction. Even when comparative plates are available and apparently progressive changes (increased opacity) appear in the later plates, one should be careful if the clinical picture is incomplete. Healing and normal repair may be accompanied by increased thickening of the mucosa and intracellular content. The interpretation of mastoid density and lack of detail as due to bone destruction is only positive when very definite. If the density increases in later plates, but the clinical evidence suggests that the mastoid is undergoing normal repair, I usually ignore the pictures unless they are so definite that there is grave danger of committing "diagnostic suicide" by doing so.

Ophthalmologist's Examination: When there is reason to suspect that a patient with suppurative otitis media is not doing well, an immediate ophthalmologic examination should be done or ordered. This examination should include taking of the fields as well as examination of the fundus and should be repeated frequently until all danger is passed. The first signs of intracranial involvement may be found in the eye. Not only that, but the first definite localizing evidence may appear there. If the first examination is negative it is a splendid record to have, for comparative purposes later, because subsequent examinations may then definitely fix the date of onset or progress of certain developments. The rapidity of progress of the process may be thus gauged and may prove to be a valuable index to the virulency of the infection.

Other important points, such as the significance of a neurological examination and internist's report, will not be discussed extensively. However, there is much more to be said about aid from other consulting specialists than space will permit. Some cases may be too urgent to permit of exhaustive and prolonged examinations, but whenever practicable it is well to get all the aid and support possible prior to operation and especially so in doubtful and borderline cases. It is much better to complete all investigations and examinations possible before operating than to operate first and then investigate. Some other condition might be found which would adequately explain some of the symptoms. It has been mentioned previously that high temperatures may occur with central pneumonias which are often so

difficult to find that only an X-ray examination will reveal them in the beginning, and that pyelitis, a frequent complication of suppurating ears in children, may be the explanation of a chill followed by temperature of 106° or higher. A meningismus with the head retracted until the occiput is against the spine between the scapulae can occur with central pneumonias, especially in children, and a real meningitis can only be eliminated in such cases by persistently negative spinal punctures. A metastatic abscess may be covered up deeply in some obscure locality, as, for instance, a large swollen liver.

Without any intention of wounding some fellow physician's feelings, I must emphasize that I have found it of great practical importance to work as much as possible with the same internist, laboratory man, ophthalmologist, neurologist or neurological surgeon in these cases. Of course, this is not always feasible. I suggest it because otitic intracranial complications are a study in themselves. It takes a great deal of experience with them to get their intricate angles and peculiarities. A neurological surgeon offers invaluable suggestions in most instances. He alone knows more about the subject than the otologist, but one should guard against allowing his own judgment to be swayed too much by an adverse opinion of almost any other consultant. A very excellent internist or neurologist may have had comparatively little experience with otitic meningitis. He may know epidemic cerebrospinal meningitis or any form of fully developed general meningitis thoroughly, but he may think entirely in terms of that completed picture. He may fail to recognize the more insidious beginning of an otitic meningitis which often lurks for days or even weeks as a small localized affair. If he does not recognize accurately what is going on, he can seriously disturb the intelligent handling of the case by a too hopeful prognosis.

Postoperative Observations: Cases that apparently do well and present no warning or danger signals until weeks after operation are very disheartening. They do occur and must be watched for constantly. As previously stated, I find them much easier to detect early and call correctly. The operative findings, having gone before, are frequently a great help. These findings should be noted carefully and accurately stored away in the mind. Mental images and impressions acquired in the operating room often help to explain what happens later or what may have begun before the operation. I have heard my associate, Dr. W. E. Sauer, stress that point repeatedly. Perhaps a small fuzzy spot over the lateral sinus, where the dura has been found attached to the inner table above, may again appear vividly in mind when the temperature shoots upward a few degrees

(without any other apparent cause) several days later. Granulations on the dura or an extradural abscess near one cranial fossa may definitely point the correct route of explortion for a brain abscess when no other localizing symptoms are present.

Many of the previously described initial manifestations of intracranial extension are easier to detect after operation than before because the physician has become better acquainted with his patient. The relative slowing of the pulse curve is one example, as a record of the normal pulse may show on the chart before the relative slowing. One also can be more positive about a change in mental attitude; it has been one of the most constant and dependable early postoperative symptoms in my experience. In addition to the mental changes already referred to, brain abscess patients sometimes begin to have gruesome dreams or nightmares, which may be misinterpreted later as actual occurrences. An alarming confusion about past events may appear. The first hallucinations and delusions are often nocturnal. Some of these patients sit up in bed, apparently half awake, with the room well lighted, and speak wildly. They get out of bed and walk about, half asleep, when they have never done so before. It may be difficult to awaken them fully or shake them out of it. This is most frightening to other members of the family as they may not be able to determine whether the patient is talking and moving in sleep or is awake and having a mental lapse. They only know it is abnormal for him. The physician may be called at three a. m. because of such occurrences, weeks after the wound has healed and the patient ha apparently recovered from his operation and has gone home. One of my patients died of multiple brain abscesses and meningitis nearly five months after the mastoid operation.

The general aspect of the patient may offer a valuable hint that he is making a losing and not a winning fight. Patients usually do astoundingly well after mastoid operations. As a general rule they recover quickly, with a daily improvement in appetite, weight, color and morale. If the patient lags in his progress and seems tired, worn or restless, if he apparently is dried out, anhydremic, and his color fades, or does not improve as it should, something is wrong. The wound may be healing splendidly and there may be no apparent reason for the lack of progress. The less evident the reason for it, the more significant it may be.

Sudden postoperative rises of temperature which cannot be adequately explained by some evident drainage block in the wound or otherwise should never be taken lightly. The better the appearance of the ear and wound, the more significant is a temperature rise.

The longer a sinus thrombosis waits, the less typical is likely to be the temperature curve. This is true usually of the onset, at least. A temperature rise may mark the beginning of a small patch of localized meningitis—a further rise may herald a slight spread or extension of the initial patch. The beginning of an area of encephalitis preceding the formation of a brain abscess may be accompanied by slight or considerable temperature.

Perhaps the first complaint may be photophobia; a very constant early symptom, which may develop later into a real cephalic pain.

Headache, whether localized or not, is unwelcome. Postoperative mastoid patients are remarkably free from pain, usually. This is often a thing at which to marvel, when much chiseling through a hard eburnated mastoid has been necessary. The pain in the head may not be connected with the mastoid region. I like it better when it is. I again emphasize that a constant headache is especially bad if its location is temporal, frontal or behind the eye and is accompanied or preceded by severe photophobia. It has also been mentioned that if the pain is neuralgic or occipital it is easier to connect up with some unevident tympanic or mastoid block in drainage, but that hope cannot be depended upon unless backed by visible evidence of such a condition in the middle ear or mastoid.

Vertigo, nystagmus, nausea and vomiting now may herald a beginning extension through the labyrinth, the progress of a brain abscess or spread of meningitis.

The precaution of having had an early ophthalmoscopic examination and frequently repeating the procedure is appreciated chiefly late in the postoperative course.

Many other interesting postoperative occurrences could be discussed except for lack of space. Detailed descriptions of postoperative developments are given repeatedly in the literature. That phase of the subject is under constant discussion and investigation. However, so much has not been written on early preoperative diagnoses.

Reference has been made already to the reasons for choosing the subject of this paper. In conclusion, it may be well to review these reasons briefly. Accurate early diagnoses of the type, location and portal of entry of beginning intracranial complications are chiefly important in otitic meningitis. Brain abscess patients may do as well when the diagnosis is not suspected until later. The present tendency is to operate as early as possible in meningitis and to wait as long as it is safe to do so in brain abscess. Nevertheless, if the diagnosis of brain abscess is made early, the case can be handled more intelligently. The patient can be kept quieter, many harmful activities

may be curtailed, and the waiting is deliberate. In any case, an early diagnosis is a distinct advantage. If nothing more is accomplished by it than keeping the patient in bed or in the hospital longer, that is something. The physician is in better standing if the sequence of subsequent events and all unfavorable developments are foretold correctly, and everything possible has been done to prepare for these things in advance. There may be little enough that one can do to alter the course of events for that matter, but whatever that little may be is done in good time. The only early therapeutic measure (if it can be classed as a therapeutic measure) in which I have much faith as a real aid is a blood transfusion. Most everything else recommended has been more or less ineffectual in my experience. I do believe in repeated blood transfusions for these early cases, and the earlier they are begun, the better.

I must admit that accurate early diagnoses have not helped me appreciably in saving lives as yet in otitic meningitis. Still, I have hopes. I can see no future hope in such cases that is not inseparably connected with, or fundamentally dependent upon, a very early and accurate diagnosis.

3720 Washington Boulevard.

MASTOIDITIS AND NEPHRITIS; CASE REPORT.*†

Dr. C. E. Kinney, Cleveland.

Nephritis as a complication of an acute mastoiditis presents some interesting points which I hope to touch upon in this short paper. The association is probably more frequent than the available literature would indicate. There are only eight cases reported as such in the past fifteen years. Shuman[1] reported one case; Sautter[2] reported three cases, Metzenbaum[3] one case, and last month Ersner and Mitchell[4] reported three more cases. Acute nephritis, particularly in children, usually follows an inflammation somewhere in the upper respiratory tract and the streptococcus is nearly always the offending organism. Scarlet fever or an acute streptococcal sore throat without rash are the two most frequent causes. If the infection involves the middle ear or the mastoid, then the question of eliminating the focus becomes of paramount importance.

Although the last word as to the pathology of nephritis has not been written, we can say that the kidney involvement in these cases is of one of two types. The more frequent is the focal or embolic hemorrhagic type. Only a few of the glomeruli are attacked, hematuria is usually the only symptom and the streptococci are easily found in the urine. The hematuria sometimes persists for months. The other type is the acute hemorrhagic glomerulonephritis in which we have most of the glomeruli involved. The symptoms here are hematuria, marked albuminuria, elevation of blood pressure, headaches, edema, uremia and death. If the patient does not expire within the first few days of the onset, the prognosis for recovery is good when the focus is eliminated.

I would like now to present the case which stimulated this paper and later discuss the points of particular interest.

On Aug. 22, 1931, a white boy, age 12 years, was seen with a left acute otitis media of twelve hours' duration with no history of antecedent upper respiratory infection or known communicable disease. The past history was negative, the tonsils and adenoids having been removed five years ago. A myringotomy was performed and

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the patient put to bed. The temperature never came below 38° C. The discharge was copious and on Aug. 27 there was enough evidence to warrant doing a simple mastoidectomy. The streptococcus hemoliticus was isolated from the mastoid. Convalescence was uneventful and there were no detectable complications.

On Aug. 28, the above patient's younger brother, age 9 years, complained of pain in the left ear. The previous history was identical with the older brother's history. The drum ruptured spontaneously five hours after the onset of the pain. A myringotomy was performed one hour later to further facilitate drainage. The patient was kept in bed and within four days the temperature curve was flat, all mastoid tenderness had disappeared and except for a decreasing aural discharge there were no symptoms. The patient was kept in bed four more days and a culture of the aural discharge revealed the streptococcus hemoliticus. During this time the child had no signs or symptoms except for the discharge. On Sept. 5, eight days from the onset, he was allowed to get up. On Sept. 6, he complained of a headache but was not seen by a physician. The next day the temperature was 39° C. and the headache was worse. Complete physical examination revealed the following positive points: Blood pressure, 138/90; slight puffiness of the eyelids and the face, moderate purulent discharge from the myringotomy wound. The urine was smoky and contained many R. B. C's. and a moderate number of pus cells. When tested for albumin the tube boiled solid. A pediatrician was called in, who corroborated the above findings. The patient was seen daily by both of us for the next seven days. The temperature averaged between 37.8° C. and 39° C. all this time and except for some accumulation of granulation tissue at the site of the myringotomy, there was no change in the ear condition. The patient became progressively more anemic but there were no other changes.

On Sept. 14, a simple mastoidectomy was done, using only nitrous oxid anesthesia. The mastoid was partially broken down, containing pus under pressure and some granulation tissue in the region of the antrum. Neither the dura of the lateral sinus nor the middle cranial fossa was exposed. Laboratory findings on that day were: Hgb. (Sahli), 52 per cent; R. B. C., 3,800,000; W. B. C., 14,500. The urine was the same as it had been for the previous eight days, except that a few hyaline and granular casts were found. On Sept. 15, a transfusion of 350 c.c. of blood was given the patient. Culture of the mastoid taken at the operation was lost. The temperature subsided by lysis, the patient's condition improved and he

was discharged from the hospital on the eighth day. Laboratory findings on that day were: Hgb., 63 per cent; R. B. C., 3,870,000; urine, albumin +, and R. B. C. + and no casts.

One week later, the urine was entirely negative and has been on several examinations since. The blood pressure slowly came down to normal, and on the twenty-first postoperative day the wound was completely healed. Examination of the blood yesterday showed it to be normal.

This was a case of acute hemorrhagic glomerulonephritis. The questions involved are several. Was this a case of scarlatina? No less an authority than Jochman[5] states that a scarlet fever kidney never starts before nineteen days from the initial onset. Did the second boy acquire the disease from the first boy? My personal opinion is that he did and that stricter precautions should be observed in handling streptococcus ears. Did we hesitate too long before operating? I believe that we did. Evidence of kidney irritation should be an indication for mastoidectomy in a case of mastoiditis. Does an anesthetic make the kidney condition worse? Sautter used drop ether anesthesia in the three cases reported without ill towards effect. With the development of nitrous oxid and ethylene anesthesia as we have it today, there should be no hesitancy from this point of view.

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10515 Carnegie Avenue.

SINUS THROMBOSIS.

Dr. Albert J. Guerinot, Pittsburgh.

The dura is composed of: *a.* Elastic tissue, connective tissue; *b.* blood vessels; *c.* nerves: *1.* sympathetic; *2.* trigeminus; *d.* endothelium.

Headache is due to increased pressure on trigeminus. Inside of dura is a layer of endothelium, which keeps pus out of the sinus, hence extradural abscess.

Arachnoid: *a.* Lamina externa; *b.* lamina interna. Between is layer of connective tissue fibres. Inside of lamina externa is an endothelial layer, hence subdural abscess.

Pia: Is attached closely to lamina interna. Space between the dura and lamina externa is subdural space. Aqueductus cochlea ends in subdural space, connects with perilymphatic spaces of internal ear. Not a physiological connection, hence spinal fluid does not go to ear. Infection can travel into ear by way of aqueduct, however, hence deaf mutism from cerebrospinal meningitis.

The arachnoidal space between lamina externa and lamina interna. Cisterna: *1.* Magna. Migraine is glaucoma (increase of fluid of the cisterna magna) because ophthalmic is located in it. *2.* Cisterna medullaris cerebelli. Goes into the petrous portion of the temporal bone, into the internal meatus. *3.* Cisterna pontis lateralis. In this lies the acousticus.

The Pacchionian bodies are granulations or excrescences on the lamina externa. Found above ten years of age. These connect with brain sinuses. Cerebrospinal fluid goes through these into sinuses of brain, and then gets into the blood.

In the dura are canals covered by endothelial layers, which are the sinuses. The circulation of blood in the sinus is irregular and very slow, especially at the periphery of the blood column.

Sinus thrombosis is produced by: *1.* Infection by contact, usually in chronic otitis. *2.* Metastatic—along blood vessels of mastoid, in chronic cases. Also in acute. High fever. May not find much but hyperemia in the mastoid process; goes along blood vessels into the sinus. *3.* Compression thrombosis; posterior fossa, extradural abscess compresses sinus, giving thrombosis. This is questionable, because there must be three factors for thrombosis: *a.* destruction

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of endothelial layer; *b.* slow blood circulation; *c.* changes in mixture of blood, chemically.

Periphlebitis does exist, hence do not have to open sinus because there is not always thrombus inside. If endothelial layer is intact there can be no thrombus. Looking at the sinus wall alone does not tell you if you have thrombus or not. The mural thrombus is first. Obstructing thrombus, spindle-shaped. Then they grow larger. Tendency to grow along large sinuses, not much in small sinuses. Grow more against brain, centrifugal, to torcular, than centripetal, towards the heart. A benign thrombus is solid, black, red, and contains bacteria. A malignant will suppurate into pus, is green, and does not show aerobic bacteria. Every thrombus is infected from a clinical standpoint, therefore remove all of thrombus.

Sinuses affected by the ear: *a.* Lateral-sigmoid; *b.* cavernous; pus found in 40 to 60 per cent of cases.

The routes of infection to the cavernous: *a.* Inferior petrosal sinus; *b.* suppuration at tip of petrous bone because cavernous sinus is right there; *c.* plexus carotidus.

Perforation in anterior inferior quadrant of drum, Eustachian tube perforation with mucus pus is not dangerous, but with creamy pus is dangerous because it goes directly into plexus carotidus.

c. Jugular bulb. More often affected secondarily than primarily.

Dangers of thrombosis: Influence the whole body by: *1.* Metastases. In brain and meninges, very dangerous. In chest: Lung abscess, bad; pyothorax, good. In abdomen, bad; spleen, articulation, joints, muscles, prognostically good.

The toxemia produces degeneration of intestinal organs and the heart. Healing of a thrombus: It tends to heal spontaneously, but patient often dies first. Wall may heal but thrombus may be inside. The wall may look normal.

Symptoms: *1.* General; *2.* brain; *3.* ear; *4.* from other organs.

1. Sensorium—physical conditions. Case has normal sensorium, in typical cases. Has slight euphoria, feels too well. Temperature, pyemic curve. An important rule is, "If in otitis media, acute or chronic, not in children, the temperature goes, without reason, higher than 102°, and then soon after to normal, suspect thrombus." If there is a chill, diagnosis is sure, even if the ear is draining free. The pulse should go with the temperature. If pulse goes down you probably have a cerebellar abscess, extradural abscess. If temperature goes down and pulse up, it is a bad sign. Chills are a bad indication.

2. Brain symptoms. Do not have focal symptoms; do not belong to sinus thrombosis. If thrombus grows in sagittal sinus may have a Jacksonian attack. May have headache in different parts of the head. Tenderness behind mastoid process suggests posterior fossa complications.

3. Eyegrounds. Not often changed. Tortuous and hyperemic veins. If you have choked disc, then go deep into the posterior fossa; may have an extradural or cerebellar abscess. The cerebrospinal fluid is normal in uncomplicated cases. If pachymeningitis interna, you may find cloudy sterile fluid. They heal nicely, if you operate radically and expose enough dura. We never operate bilaterally in one session; means double mastoid. One side is usually worse. Operate on worse side, then if you suspect complications operate again on the same side.

Ear symptoms: Acute otitis. Mastoiditis needs three weeks for development. Sinus thrombosis may develop in four days. Acute exacerbation of a chronic otitis gives intracranial complications. Ear may clear up and be dry, and then thrombosis may occur later, may have a chill.

4. Other organs: Blood findings: Negative does not mean much. If taken between two chills may be negative. Positive of value when you have sepsis with diminution in resistance of patient. Leukocytosis more important than bacteriological. Tumor of spleen. Subicteric injection of conjunctiva.

Differential diagnosis: *a.* Central pneumonia; *b.* gastrointestinal catarrh; *c.* latent appendicitis.

Atypical otitis in children and adults over sixty may get chills and fever high; do a paracentesis. If it is just an otitis, symptoms will clear up; if not, you have a thrombus.

Differential diagnosis; incomplete operations: Cholesteatoma — simple operation. In convalescence you get fever and chills. If you get fever, complete operation, else you get sinus thrombosis or meningitis. If you expose dura and close wound, may get fever; open whole wound. After exposing dura, first dressing on fifth day. If you get slight fever early, do dressing and open wound.

Tonsillar disease, with running ear. Up and down temperatures. Big point is irregularity in secretion. In morning much, at night very little, next day the same, bad symptoms, possibly means sinus trouble.

Infectious diseases: Scarlet fever, measles, with running ear and questionable exanthem, operate ear if secretion is irregular.

Wound infections: Edge red. Use antiseptic solution: aluminum acetate, Burrow's solution. If this does not clear up, you may have metastatic condition in the skin; cellulitis, no pus. Wound infection lasts two or three days. If it grows in spite of treatment, it is metastatic. If this is so, the prognosis is bad. Opening the skin does no good.

Brain diseases: Do not have to be differentiated because operation is needed, as meningitis, brain abscess, etc.

Real pneumonia: Irregularity of secretion. If it has signs of a mastoid or sinus thrombosis, operate fast.

Prognosis depends on: *1*. Time you operate; earlier the operation, the better the prognosis. *2*. Condition of the heart and blood vessels. Bad if lues, sclerosis or alcoholism. After sinus thrombosis operation the temperature should go down slowly. If it goes down quickly, means heart collapse. *3*. Findings at operation. The more you find, the better. Big thrombus is a good sign. Bad if you have up and down temperature but no thrombus. If they have metastasis, it is good sign; not, however, if it is in brain or abdomen. *4*. Kind of otitis. In the acute type the prognosis is better. *5*. Kind of operation. Operate at once radically; 20 to 25 per cent mortality.

Treatment: Divide the cases into two groups: *1*. Diagnosis is made clinically first. *2*. Sinus thrombosis found at operation.

1. Start with jugular ligation. Why? *a*. Then you can operate aseptically. *b*. Work with chisel is dangerous; makes concussion; parts of clot may be pushed into blood stream. If vein is closed, the chief way for emboli going to body is shut. Incision made in middle third of vein is easiest. Not necessary to tie above facial vein. Resection is made only if there is edema around and thrombus in jugular vein. Resect below thrombus. If thrombus goes way down into subclavian, do not ligate but just cut into vein. Take out what you can.

In ligation, allow silk to hang out of wound because sometimes it is necessary to make jugular skin fistula. If bulb has a thrombus, pull out one end of vein, remove tie, and sew it into skin and you have natural drainage for the bulb. You can do this twenty-four hours after sinus operation. If too much bleeding, tie off and then forty-eight hours later operate again. Closes in about two days if there is no secretion. Operate then on the ear.

Expose sinus wall fully till you get normal sinus. It is only necessary that you get normal wall against the brain. It is not necessary to have good wall down to the bulb. Puncture sinus with knife.

Do not stop operation until you get free bleeding from brain area. Not necessary to have free bleeding from the bulb. When there is free bleeding, stop it with iodoform gauze.

2. Cases diagnosed at the operation: Stop ear operation. Do jugular ligation, then go back to the ear. Alexander avoids jugular in patients over sixty who have had only one chill. The dressing is made on the second or the third day. Take tampon out and if bleeding, tampon it again. Then change dressing every day.

Conservative Treatment Following Operation: 1. Alcohol, much wine to drink. Children get alcohol. *2.* Blood transfusions, in cases in which it seems all is lost. (If after sinus thrombosis operation there are chills and fever again, suspect thrombus in bulb.)

604 Westinghouse Building.

VON RECKLINGHAUSEN'S DISEASE, WITH REPORT OF A CASE INVOLVING THE EXTERNAL AUDITORY CANAL.*

DR. HENRY S. WIEDER, Philadelphia.

It is with some hesitancy that one presents the subject of von Recklinghausen's disease before a body of otolaryngologists, as it is a condition which concerns particularly the dermatologist, surgeon or neuropathologist, but the recognition of some of the rarer diseases in the other fields of medicine is often important even to the otolaryngologist, especially when they occasionally involve his field.

Von Recklinghausen's disease or multiple neurofibromata, is an affection primarily involving the endoneureum of the nerves, principally the vagus and the other cranial nerves, and especially the peripheral nerves. It assumes various phases or forms, which can grossly be summarized under four heads: generalized neurofibromatosis; neurofibromatosis of skin, also known as molluscum fibrosum; plexiform neurofibroma; and elephantiasis neuromatosa.

Neurofibromata was first recognized in 1793 by Tilesins and was called neurome in 1803 by Odier. Discott in 1813 described multiple nerve tumors, and Bruch in 1847 suggested that they were due to polypoid growth of the neurolemma of the nerve. Verneuil in 1857 described the plexiform neuromas. In 1863 Virchow gave a lengthy description of these tumors, dividing them into true, mixed and false neuromas. Von Recklinghausen in 1882, in an exhaustive review of the subject, grouped all the conditions under one head, multiple neurofibromata, including the multiple tumors of the skin, known as fibromamolluscum. He held that the majority, if not all, spring principally from the connective tissue sheaths of the nerves, afterwards spreading upward along the nerves, vessels, coil glands and follicles in the skin. His findings were confirmed by Kriege and Unna. According to Unna, "the subcutaneous nodules show a distinct plexiform arrangement and consist of cords of finely fibrillated transparent cellular connective tissue. Medullated and nonmedullated nerve fibres run through them, rarely united, but usually widely separated from each other. By the growth of the inner coats, the outer layers of the nerve sheaths are enormously dilated. A peculiar

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variety of the mast cells is the most striking characteristic. They are distributed throughout. Some of the mast cells are surrounded by a red halo, about the size of the ordinary mast cell. This halo shows the same red color as the granules but it is not granulated material, but spongoplasm."

As stated before, the disease manifests itself grossly in four different forms, but the underlying pathology in all these forms is identical, namely, connective tissue hyperplasia of the endoneureum of the finer nerves, separating the individual nerve fibres or shoving them to one side. This hyperplasia is not uniform but attacks the nerves to a different degree in various portions, so that it gives them a beaded appearance. In some areas it results in marked dilatation and more or less localized tumor formation. In other areas it assumes merely the form of an irregular enlargement of the nerve as a whole. Sometimes the nerve can be traced out by the nodular sensation imparted to the palpating hand. This connective tissue is usually rather soft, sometimes almost myxomatous in character. In other areas it may be quite firm. When there are a group of nerves involved in close proximity to each other we have produced a plexiform neuroma or "Rankenneurom" of Bruns. These are more or less localized pendulous tumors that impart the sensation similar to that felt on palpating a varicocele, namely, like a bundle of worms. They sometimes attain a considerable size. They are prone to be found on the head, temple and upper eyelid, frequently on the posterior part of the neck behind the auricle, occasionally on the nose and cheek, and sometimes on various other parts of the body. The component parts are loosely held together by connective tissue.

In elephantiasis neuromatosa, the skin and subcutaneous tissue is enormously thickened, so that it resembles elephantiasis due to infestation by the filaria. Its origin, however, is from the endoneureum, but it differs from the other types of neurofibromatosis, in that the connective tissue invades the surrounding structures, causing the great increase in thickness of the subcutaneous tissues. It replaces the normal cutaneous and subcutaneous fat more or less completely. In areas the connective tissue presents a watery, glistening, grayish-white appearance. Some parts are soft and gelatinous, others are drier and firmer. At times fibrous nodules and strands may be felt in the midst of the swollen tissue. These represent the affected cutaneous nerves as in generalized neurofibromatosis.

The fourth and final form of the disease is the form with which we are most concerned. In this condition the surface presents anywhere from a single one to two thousand tumors, varying in size

from the head of a pin to the size of a child's head. One tumor weighing forty pounds has been reported. These growths may be round and pedunculated or be hemispherical and sessile. Some project beyond the surface, while others are distinctly subcuticular, so the skin can be rolled over them freely. Some are not visible but can merely be felt by rolling the skin over the surface. They impart a sensation of grains of sand beneath the skin. Some are extremely soft and almost difluent. By pressure they can be made almost to disappear, only to reappear on release of pressure, as is the case in palpating a hernia. Others are quite hard, while still others have a hard external portion with a soft center. All portions of the cutaneous surface may be affected, and even the mucous surfaces, such as the cheeks, lip, palate, and tongue, and even portions of the gastrointestinal tract. The skin covering the smaller ones is usually pale, while over the larger ones it is often bluish-red and tightly stretched, with many blood vessels and hypertrophied hair follicles. Occasionally they are inflamed or even ulcerated. They are rare upon the soles of the feet and palms of the hands, being flattened out when in those situations. The growths contain a large amount of serum and lymph and are prone to be very vascular, giving rise to rather profuse hemorrhage on their removal. They are very frequently associated with pigmentary deposits in the skin, either over the growths or separate from them. There appears to be a marked tendency to the sarcomatous change in these growths, but it partakes of a different nature from the sarcomatous change in the true neuroma. It does not show the same tendency to generalized metastasis or general sarcomatosis. It is likely to recur in the stump of the original nerve affected. In very late stages of the disease it may involve the internal organs. It is, however, eventually as fatal as other types of sarcoma. Sarcomatous degeneration may be precipitated by trauma, especially of the operative type when the operation is incomplete. Trauma or incomplete operation also has a tendency to stir up the activity of otherwise stationary tumors.

Little or nothing is known as to the etiology of this disease. The number of cases reported is not very abundant, but among them there is shown a considerable tendency to heredity. In three instances two brothers were affected and quite a few instances either the parents or grandparents were affected with some variety of the disease. It may be present from birth, but because of the nature of the disease involving the nerve trunks without symptoms, and because of its slow development, it may not be manifest until later in life, especially at puberty. It peculiarly enough is associated frequently

with other defects of the body, mental retardation is a more or less common occurrence and pigmentation, either localized or more or less general, is a very frequent accompaniment. As a matter of fact, the pigmentation often precedes the external manifestation of the neurofibromatosis.

As to the origin of this disease, little is known. It is a disease of the peripheral nervous system exclusively, all portions being subject to the disease, namely, the cerebrospinal nerves, the sympathetic nerves, and the nerve terminations in the skin. The central nervous system has never been known to be attacked, although the disease may be associated with concommitant tumors or affections of that system. Peculiarly enough, while the coverings of the brain and spinal cord are considered analagous or identical with the coverings of the nerves, they are exempt from this disease.

There is no indication that the condition is due to any external influence or any intoxication or infective process. The condition seems to run a certain course, reach a certain development and often stop in its growth. As stated before, the growth may be accelerated or renewed as a result of traumatism. Von Recklinghausen pointed out that these skin tumors were most numerous where the most friction and pressure were found while their absence from the palms and soles is attributed to the protection given the nerves by the thick pad of fat present in these situations. In considering the symptoms associated with this disease, we find that commonly there are none, although we may have pain, parasthesia, anesthesia or weakness. Joint changes due to atrophic changes have been described, also scoliosis has been found in a few cases. Seefelder called attention to the hydrophthalmus present in these cases. These growths are not usually sensitive, excepting those growths which are subject to pressure or injury. They are usually freely movable and painless unless undergoing malignant changes. The nerve fibres and functions of these fibres are usually not affected, although degeneration of the nerves has been described in a few cases. When tumor-like enlargements of the nerve roots occur within the spinal canal we may have symptoms from pressure on the spinal cord resembling the symptoms of compression of the cord, due to other causes. Motor symptoms are much more common than sensory symptoms. While the cranial nerves are often affected, especially the vagus, it does not commonly give rise to symptoms, except in those rare cases originating close to the brain, in which case they simulate an intracranial tumor. Involvement of the sympathetic nerves, while often extensive, rarely gives rise to symptoms, unless it be failure of

nutrition in the later stages of the disease and the susceptibility to intercurrent diseases, such as influenza, tuberculosis, etc.

Prognosis: Patients affected with neurofibromatosis may live to a ripe old age, the tumors attaining a certain growth and then becoming stationary. The chief cause of death in cases in which it is attributable to this disease is sarcoma. This may occur without any apparent cause and is more common after injury. In some cases, when the disease has been progressive in character and the whole

peripheral nervous system becomes involved, the patients appear to pass into a cachectic state with loss of weight and strength, anorexia, apathy, listlessness, loss of memory and other mental faculties and death from general exhaustion.

The case I wish to report is of interest because of the involvement ment of the external auditory canal by one situated within the concha of the ear. This growth gave the impression of being a cystic growth or a lymphangioma. The history is as follows:

R. G., age 31 years, white female, gave a history of being cognizant of a growth obstructing the left external auditory meatus for about eight years, but did not know how much longer it may have been present. She said as long as she could recall the hearing in her left ear had been dull or obstructed. She first noticed it when she had her hair bobbed. It had never given her any pain until the last two months, when she noticed some discomfort in the left ear. The patient has numerous small growths of various types over the rest of her body, some being pedunculated, soft fibrous; others being sessile, almost semifluctuating masses. She has marked scoliosis and scabard tibae. She was admitted to the Graduate Hospital under Dr. Robert's service on June 29, 1931, desiring to have the growth in the left ear removed.

Examination showed a soft mass about 2½ c.m. in diameter, completely occluding the external auditory meatus of the left ear and attached over half its circumference to the concha. It extended down into the external auditory canal to the face of the tympanic membrane, which was seen only with great difficulty after pushing the growth to one side. By insinuating the little finger into the external auditory canal the mass could be almost obliterated. In the scalp was another growth, about 5 c.m. in diameter, irregularly round, which, when pressed in an upward direction, could be almost obliterated but would refill when pressure was released. The patient said at this time she would hear "sort of a peculiar noise in the ear." X-ray of the skull showed asymmetry in development, the left mastoid tip being completely absent. The region of the antrum and petrous bone were increased in density. The face showed hemiatrophy and asymmetry, the lambdoidal suture on the left side was not closed. Dr. Cohen, the Roentgenologist, seemed to feel that we were dealing with a lymphangioma or possibly some atypical form of meningocele communicating with the cerebrospinal system, as the growth could be almost completely obliterated by pressure and would promptly refill. Examination was made by Dr. Greenbaum, who gave the opinion that we were dealing entirely with a case of von Recklinghausen's disease (Molloscum fibrosum), with tumors in various portions of the body, including the external auditory canal. This opinion had been also expressed by Dr. Walter Roberts and was confirmed by puncture of the intra-aural growth and the growth in the scalp, both of which failed to yield any fluid. The Wassermann test was negative.

X-ray taken by Dr. J. G. Cohen on July 1, 1931, revealed the following: "There is maldevelopment of the left mastoid with considera-

ble atrophy of the osseous structures. The tip is practically unformed. There is increased density in the middle ear structures and toward the apex of the petrous portion of the temporal bone, extending in almost to the internal auditory meatus. Accompanying these changes in the mastoid and the petrous portion of the temporal bone, there is definite atrophy with sclerosis of the mucoperiosteum of the left maxillary sinus. The mandible is also smaller than normal for a skull of this size and on the right side is the seat of some cystic-like degeneration. In addition to this the molars are malformed, the left third molar impacting the second, distor-horizontally. The teeth are quite carious and there is a large cystic abscess present involving the apices of the left central and lateral incisors. There is also an unerupted, deciduous, supernumerary left third molar. There is a rather localized density present over the lambdoidal suture. These findings suggest the probability of the connection of this cystic tumor mass with the cerebrospinal fluid pathways, possibly interconnected through the lambdoidal suture. The base of the skull is definitely shortened, giving the skull a pseudodolichocephalic appearance. This is undoubtedly the result of the presence of mature synostosis of the basil structures. The anterior and middle branches of the middle meningeal arteries which are also exaggerated on both sides."

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2106 Spruce Street.

OTOLOGICAL SEPSIS FOLLOWING SUBMUCOUS RESECTION OF THE NASAL SEPTUM.

Dr. Louis C. Menger, Brooklyn, N. Y.

This patient was admitted April 23, 1930, to the Otological Service of the Bushwick Hospital. He had suffered from nasal obstruction and frontal headaches for four months and because of these complaints, he consulted a rhinologist, who recommended and performed a submucous resection of his nasal septum two weeks previous to his admission to the above hospital. Three days following this operation he began to feel ill, suffering from occipital and frontal headache, and at this time his right ear began to discharge purulent material. There had been no pain in the affected ear at any time and as far as he knew he had never had a discharge from the same. His physician advised him to take a vacation in the country, to improve his physical state, and he accordingly decided to motor to Liberty, N. Y., driving his car himself. Upon his arrival there he felt quite exhausted and suffered a severe chill. There was no pain present in his ear but the discharge still persisted and he noticed that he was becoming deaf on the right side.

He was removed to a sanatorium in Liberty, where he remained four days. The first day of his stay there showed an evening temperature of 103°. The following morning he had a chill, and a temperature of 99°. In the evening his temperature rose to 105°. The next morning he had another chill, and a temperature of 100°. The evening of the same day his temperature again went up, reaching 104.8°. The next morning he again had a severe chill, his temperature registering 100.6°. His pulse and respiration rate was in accordance with the temperature. After the last mentioned chill he was advised to return to New York for hospitalization there. The rest of his history bares no significance to his ailment, and is therefore omitted.

His admission to the Bushwick Hospital on April 23, 1930, showed a well developed, white male, age 33 years, acutely and critically ill. He was very drowsy, his temperature 104°, pulse 110, respirations 30, tongue heavily coated. There was a nonodorous, purulent discharge in the right canal. The drum had a perforation in the pos-

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terior fold which was amply draining the tympanic cavity, there being no bulging of the drum present. There was no swelling or tenderness over the right mastoid region. The canal wall was normal throughout. His nasal examination showed some difficulty in breathing through the nose, the scar of a recent septum operation on the left side, a large amount of mucopus on the left nasal floor. The frontal sinuses did not transilluminate. His right antrum was clear, the left did not transilluminate. There were no signs of meningitis present. Eye examination showed negative findings, except some fullness of the retinal veins. Heart and lungs were negative. Abdomen showed tympanitic distention, spleen was palpable, liver not palpable. Blood count, 13,200 whites, with 76 per cent polys.; 4,920,000 red cells, 78 per cent hemoglobin. Urine, 3+ albumin; faint trace of sugar and a few pus cells.

Our impression at this time was that we were dealing with a possible blood stream infection from the ear or nose. A sinus and mastoid X-ray study was made the following day, the report of which was: Right mastoid cloudy with cavitation, undeveloped frontal sinuses and a cloudy left antrum. The positive X-ray findings were noted, but it was assumed that the patient was suffering probably from some intracranial complication, the most likely of which was a sepsis originating from the lateral sinus, and it was thought that further study would be wise in order to determine what type of operative procedure would yield the best results. We felt at this time that a simple mastoid operation would not be sufficient to yield the desired result and, therefore, to effectively deal with the situation before us, he was kept under observation, bearing in mind the facts that he had recently undergone a nasal operation and also that his aural disease was a likely potential source of his sepsis.

On his second day in the hospital his temperature was 104.8° at 8. a. m., dropping to 102.2° at 4 p. m. At this time he stated to us that he felt unusually well and sat up, supported by a back rest, in bed to read. To add to our knowledge of the case, a spinal tap was done, withdrawing 6 c.c. of clear fluid, not under pressure, showing 60 cells per c.m. Unfortunately, by the time the technician did the differential count, the type of cells was not possible, owing to a disintegration of the same.

Blood cultures, several in number, were taken from the first day of his stay in the hospital, and were all repeatedly negative. His urine constantly showed albumin, without any other findings of importance, until his temperature became normal later in the course of the disease.

On the third day in the hospital his low morning temperature was 99.2° and his high temperature was 101.6° at 3 p. m. He offered no complaints, his tongue appeared to be clearing, he took freely of soft food and in general seemed to be making favorable progress. At this time he developed an occasional cough and expectorated thick, tenacious mucus. This sputum was examined by the laboratory but showed nothing that would be of any diagnostic importance. The chest examination was also negative.

Red and white cell counts and hemoglobin estimations were done daily and remained about the same as stated in the report previously submitted.

On the fourth hospital day the a. m. temperature was 101°, pulse 70, respirations 25. There was a moderate, purulent, aural discharge, right. No local signs of mastoid involvement. His nasal breathing was improved and there was less discharge on the left nasal floor. He told us, at this time, that he felt better than he had for weeks. In the afternoon, at four, his temperature went to 104°. Although he had no complaints, he became somewhat drowsy. As previously stated, the blood cultures taken at the height of temperature were all negative; his red cells and hemoglobin showed no change. At 10:45 p. m. his temperature went to 106°, with a pulse of 100 and respirations of 25. He complained of severe drawing pains in both legs, and became very drowsy. In general his condition seemed much worse. We decided that further temporizing would result in the patient losing ground and concluded to uncap the right mastoid and expose and open the lateral sinus, as this seemed to be the most likely source of his sepsis.

He was taken to the operating room at midnight and, under gas-oxygen-ether anesthesia, the usual incision back of the auricle was made. This was joined by an incision at right angles to the same over the horizontal portion of the sinus later. The cortex was moderately thick and very hard; the mastoid structure consisted of small and moderate size cells. These cells contained pus and granulation tissue. Bony softening was encountered around and below the antrum, and in the region of the descending portion of the lateral sinus; this area of the sinus dura was covered by minute granulations. The sinus was uncovered from the knee to near the bulb, and plugged in the usual manner. There was free bleeding from both ends, after incising. The opening in the sinus was packed with plain gauze; the horizontal incision and the upper half of the wound was closed with silkworm sutures. The jugular vein was isolated and resected above the facial.

First day postoperative: Patient showed a good reaction to the operation, but continued to be very drowsy. Low temperature, 98.6°; high temperature, 101°. He was transfused with 500 c.c. of whole blood.

Second day post-operative: A short chill in the afternoon, followed by a rise of temperature, reaching 105.2° in the evening. At this time he complained of severe temporosphenoidal headache. The only objective sign of meningeal irritation was a slight neck rigidity. Another lumbar tap was done, with practically normal findings.

Third day postoperative: Low morning temperature, 103.2°; high evening temperature, 106°. Nurses' reports showed he had experienced a restless and sleepless night. His drowsiness persisted. The neck and mastoid wounds were inspected and appeared clean. An eye examination showed: Pupils small, sluggish reaction to light; later examination under a mydriatic showed a clear media, with fundi essentially negative; there was no evidence of increased intracranial pressure.

Fourth day postoperative: Low morning temperature, 104°; high evening temperature, 105.6°. Has had another restless, sleepless night. Patient very irritable and complained of pain over the right eye.

Fifth day postoperative: Low morning temperature, 102.2° at 4 a. m., rising to 103.6° at 8 a. m., dropping in the evening to 101.4° at 8 p. m. The left antrum was punctured and irrigated and showed no mucus or pus in the return. In the afternoon he appeared to be improving; his tongue began to clear, his drowsiness was considerably less and, in general, his condition became more favorable.

Sixth day postoperative: Patient had had another restless night. At 5:45 a. m. he had a chill lasting four minutes. Early morning temperature was 101.4°. His general condition was not as good as on the previous day. Slight neck rigidity again elicited. Another lumbar tap showed twenty cells, with 51 per cent polys. and 49 per cent lymphs., sugar a trace and the slightest possible trace of globulin. The fluid was cultured but showed no growth. The sinus plug was removed at this time with some moderate free bleeding. Neck wound inspected and proved to be clean. He was given 250 c.c. of whole blood intravenously. One hour after the transfusion he had a chill lasting ten minutes. He became irrational during the night, but was perfectly rational the following morning.

Seventh day postoperative: Low morning temperature, 101.2°. High evening temperature, 101.4°. Slight neck rigidity still present. Lips dry, tongue coated. Mastoid wound clean, neck wound healed.

Eighth day postoperative: Low morning temperature, 100°. High evening temperature, 105°. General condition seemed good. Patient perfectly rational. Neck rigidity seemed more marked than on previous examination.

Ninth day postoperative: Low morning temperature, 99°. High evening temperature, 102.2°. Patient bright and cheerful, tongue still dry, but was beginning to clear in the center.

Tenth day postoperative: Temperature began to decline. On the following day it rose to 103°, but the next day came down to near normal. He then ran a low grade temperature until the twenty-eighth day postoperative, when it became flat normal and remained so until he left the hospital, after having been there thirty-six days.

With the decline in his temperature practically all of his symptoms disappeared with the exception of an indurated swelling, which showed in the neck on the sixteenth day postoperative below the tip of the right mastoid, posterior to the sternocleidomastoid muscle. This made him very uncomfortable for about a week. It was an infected lymph node and looked, at first, as though it was going to break down, but local measures, plus a therapeutic dose of X-ray, seemed to exert a favorable influence, a progressive improvement and ultimately resolution taking place.

This patient when last examined, on Dec. 3, 1931, stated that he had been in normal health, strength and weight since he left the hospital.

It was somewhat difficult at first for us to decide where the sepsis was coming from; whether from the nasal path or the mastoid. Of course, we assumed that the middle ear infection was secondary to the submucous resection *via* the Eustachian tube, but with symptoms suggesting a blood stream involvement coming on directly after an intranasal operation observation from this angle was warranted: there was no evidence of a septal abscess present but the possibility of a thrombophlebitis of the septal veins was to be considered, although there were no local findings to help us make this diagnosis. As events developed, our attention was therefore focused upon the otological picture and our therapy directed in this direction, with an ultimate favorable outcome.

202 Weirfield Street.

PLASTIC SURGERY OF SADDLE NOSE.

DR. M. WILLIAM MOOTNICK, New York.

Saddle back nose is an important and rather common condition of the nose coming to the attention of the plastic surgeon. Saddle noses are either congenital or acquired. The congenital are syphilitic and nonsyphilitic. The acquired are due to trauma; septal abscess; following faulty nasal septum operation; ozena, with destruction of the nasal bones; gumma, destroying the nasal bones; and tuberculosis.

The avenues of entrance of implant to the seat of deformity are five, the choice of which depends mainly upon the internal condition of the nose. Where there is no pathological condition within the nose, the route of choice is the standard incision, also known as the endonasal route, in which a transverse incision above the lower border of the triangular cartilage of the nose is made. On the other hand, if a pathological condition exists it should, if possible, be treated and eliminated, in which case the external route is used as the avenue of entrance.

The external routes are described here in their avenues of choice. Firstly, a transverse incision below the nasal tip end. Secondly, a vertical incision into the columella. Thirdly, an incision over the inner one-third of the eyebrow. Fourthly, a transverse incision at the infraglabellar region.

Materials used in implants in saddle noses are of many varieties. They are cartilage, bone, fascia, ivory (elephant tusk), walrus tusk, vegetable ivory, synthetic ivory imitations, gold, celluloid and paraffin. All of these have been used for prosthesis in saddle nose and for other plastic operations on other parts of the body.

Cartilage: Autoplastic costal cartilage is the most favored and approved material from the standpoint of ultimate healing and organization. Complications following its removal, however, may be serious, as injury to the pleura, and possible infection and empyema. One other objection to costal cartilage removal is that it requires an extra operation, which plastic patients are loathe to undergo, as the patient would be confined necessarily in bed with symptoms of a fractured rib for a considerable length of time.

The rib, from the sixth to the twelfth, depending upon the size required, is removed either with local or general anesthesia. As a note of importance, in removing the cartilage from the rib, do not

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remove its entire thickness, but always leave the rib in continuity. The cartilage is then shaped with a knife, making straight cuts without bending it. The perichondrium must be left always on one side of the rib. Curling of the cartilage towards the perichondrium is due mainly to faulty technique.

To prepare the nasal deformity for reception of the cartilage implant in cases with no intra- or extranasal pathological conditions, the endonasal route is the one chosen; and, if otherwise, the external route is used.

For the endonasal route operation, aseptic and antiseptic precautions are taken inside and outside of the nose, vestibule and face by washing with tincture of green soap, following with bichlorid of mercury 1:1000, and, lastly, by 70 per cent alcohol. The nose is irrigated internally with normal saline solution; while the posterior nares are packed with iodoform gauze. Afterwards, the cartilage is shaped to its proper measurements to fit perfectly in its new anatomical position. It must be remembered that from the time the costal cartilage leaves the chest wall and until it enters the bed of the nasal deformity, it must not be touched by the hands. It is held with sterile gauze while shaping with a knife, and must be inserted in its bed with a sharp-toothed anatomical thumb forceps. Touching the wound edges must be avoided by the use of metal retractors. The wound is closed with a few fine silk sutures, the tying of which must be accomplished with two pairs of thumb forceps. The sutures are removed after three or four days. In cases of marked defect where a large, thick and long piece of cartilage is required to satisfy the contour of a good dorsum and lateral walls, the costal cartilage obtained will be found to be insufficient. However, costal cartilage is ideally suited for medium-sized defects. In small bridge defects the cartilage obtained from the auricle or from the lower lateral cartilages of the nose are sufficient. This requires only a minor extra operation.

Fascia Lata: Fascia lata has been used successfully for filling in defects in saddle back nose.

Bone: Bone removed from the anterior crest of the tibia. With bone implant it is absolutely necessary to include its periosteum, without which it would absorb rapidly. Even with its periosteum the bone shrinks, so that an original larger piece is required, giving, therefore, a poor postoperative cosmetic appearance. Bone is not good material to handle and to shape; causes too much trauma to its surrounding tissues; and in healing does not give a good cosmetic result, due to occasional improper or faulty healing. The healing of bone transplants is not as good as of cartilage. When a bone implant

is infected, it will surely be expelled, while cartilage or ivory still may remain *in situ* with proper postoperative treatment.

Other disadvantages are a fracture of the tibia, following a sudden misstep; infection; osteomyelitis. Bone procured from rib, and bone plus cartilage from the rib have the same disadvantages. Bone from

Fig. 1. Fig. 2.

Fig. 1. Case 1. Saddle nose following septal abscess.
Fig. 2. Case 1. Corrected by costal cartilage.

Fig. 3. Fig. 4.

Fig. 3. Case 2. Saddle nose following faulty resection of septum.
Fig. 4. Case 2. Corrected by ivory implant.

the middle turbinate is very difficult to render sterile. It is not as suitable as cartilage or ivory.

Paraffin: Paraffin did not prove successful, and is now discarded because of its dangers and disadvantages. Paraffin was used by subcutaneous injection with a special syringe at a certain degree of temperature, and was molded into its proper shape by the operator's

fingers. The paraffin was used in three grades: soft, hard and mixed. The dangers of paraffin injections are embolism, causing blindness or sudden death; paraffinoma; necrosis; and infection. The infection in this case is very severe, refractory and difficult to control. In saddle nose a large amount had to be used at the base in order to get a narrow crest or dorsum. Eventually, there followed often gravity dislocation with final poor cosmetic result.

Gold and Celluloid, these are inorganic substances. They have been used successfully by some operators who maintain good results in this and other fields of plastic surgery; yet they act as distinct foreign body irritants, and are not to be used.

Walrus Tusk, vegetable ivory or any of its imitations are not tolerated by the body tissues, and should not be used.

Heteroplastic Ivory: Next to cartilage, ivory obtained from elephant's tusk is best suited; is nonbrittle; nonabsorbable; easily sterilizable; and insures permanency and firmness by its becoming firmly encapsulated in its new anatomical relationship. Ivory is an organic substance very similar to human bone; easy to handle and to shape; is easily obtainable; and effectively sterilized by boiling water. Dr. Jacques Joseph, of Germany, is a great advocator in the use of ivory for rhinoplastic prosthesis. The most suitable ivory is that obtained from the tusk of a healthy adult elephant. Genuine ivory does not change its color in sulphuric acid. There are three ways to prepare the ivory. The one is its preparation prior to operation; the second is at the time of operation; and the third is the combined method, *i. e.,* shaping the ivory before and during the operation.

Principles in Shaping the Ivory: The implant must be adapted to its new anatomical bed in the nose. The anterior surface of the ivory must be shaped in conformity to the normal sculptural characteristics of the nose. The lower end of the ivory must blend with the nasal tip, while the upper end must blend with the glabella. The lateral walls of the ivory must blend with the lateral margin of the saddle nose. The length, width and depth measurements of the seat of the depressed deformity are marked upon the block of ivory to be used. The upper end of the ivory is wedge-shaped; the lower end of its posterior surface is placed between and above the crus apicis cartilage, and is concave from below upward and concave from side to side, and separated in the middle by a ridge concave from before backwards. This ridge will occupy a slit between the two apicis cartilages to secure a proper sitting of the ivory. Immediately above this posterior concavity, which corresponds to the deepest portion of the saddle, and at that point the ivory is thickest and occupies

about one-third of its middle posterior surface. From there on the ivory continues in a more or less incline in accordance with the contour of its bed, thinning and tapering towards the glabella where it meets the anterior surface, thus completing the wedge of its upper

Fig. 5. Fig. 6.

Fig. 5. Case 3. Slight saddle nose following faulty septum operation.
Fig. 6. Case 3. Corrected with ivory implant.

Fig. 7. Fig. 8.

Fig. 7. Case 4. Front view. Congenital (nonluetic) extreme saddle nose.
Fig. 8. Case 4. Front view. Corrected with ivory implant.

ivory extremity where it blends in with the root of the nose, or glabella. Always round out the sharp points and edges of the ivory. In cases where the nasal septum protrudes slightly at the base of the saddle, a groove should be made on the under surface of the ivory

to allow for the protrusion. The shape of the implant must fit exactly into the deformity. All this is done with a rough emory wheel, metal saw and with different rough and fine metal files. No casts as in costal cartilage or plaster of paris masks are necessary for success. Ivory is sterilized by boiling in water for one hour.

Fig. 9. Fig. 10.

Fig. 9. Case 4. Profile. Condition as presented.
Fig. 10. Case 4. Profile. Reconstruction.

Fig. 11. Fig. 12.

Fig. 11. Case 5. Slight saddle nose, result of faulty septum resection.
Fig. 12. Case 5. Corrected by auricular cartilage.

Endonosal (Vestibular) Operation: The route of access in this operation is on one side of the nose only, the left side preferred. Make about a one-half-inch incision transversely above the lower margin of the left triangular cartilage, not too near the septum. Separate the skin by undermining it over the bridge of the nose with

a double-edged straight knife, curved slightly in its long axis. Separate also the skin over the depression; then use a left comma knife to separate the skin over the crus apicis and lateralis down to the nasal tip. Do not undermine the entire skin covering of the nose but enough to make a comfortable bed for the piece of ivory. Avoid cramping and tension on the skin above. At the same time do not allow the skin to be too loose over the ivory. By adhering to the above technique a kind of pocket is formed for both extremities of the ivory piece. This pocket will keep the ivory in place securely. The sterilized ivory is then grasped with a heavy, sharp-toothed anatomical forceps, and placed through the original incision into the space of the deformity, and if the ivory is a perfect fit, it will just

Fig. 13. Fig. 14.
Fig. 13. Case 6. Saddle nose following trauma.
Fig. 14. Case 6. Corrected with ivory implant.

fall in, and settle into its new bed as if it belonged there. Care must be taken that the ivory is not handled, or the edges of the wound touched with it while straddling the ivory over the seat of the deformity. If, accidentally, the ivory becomes unsterile it must be resterilized in boiling water for a period of five to seven minutes. The wound is then closed with two or three fine silk sutures, size No. 1, and not too near the implant. They are removed after three or four days. In the event of an additional deformity, as where the nasal tip has no support from below and is more or less flat against the upper lip, the avenue of incision in this case would be the external route, or columella incision (Dr. Sheehan). This incision begins a little below the nasal tip and extends to the philtrum. The ivory is

prepared in the same way as mentioned in the saddle back operation, with the exception that a perforation is drilled through at the lower end of that part of the ivory which rests over the crus apicis cartilage in order to allow the specially prepared upper end of a thin strip of ivory, which is placed within the columella and which extends downward and rests at the anterior nasal spine. This joint which unites the two implants allows the nasal tip more motion and flexibility when the nose is later handled by the patient. When one wishes to correct the two defects, namely, the saddle back and the depressed nasal tip, with an implant made of one piece in the shape of a solid angle, there

Fig. 15.

Fig. 15. Case 6. Roentgenogram showing the ivory prosthesis.

follows only limited flexibility of the nasal tip. Do not suture too near the implant. Splints, apparatuses, bandages to retain the implant in place are unnecessary, as the properly fitted implant retains its place without any difficulty. It is advisable to soak the ivory in cold water overnight so that it will be softer in consistency and easier to shape. In some extreme cases of saddle back it is necessary to build up the nose before the deformity is measured. The building up is done by raising, if necessary, the nasal tip, and by narrowing and raising the bridge, if possible, before measuring for implant. If the crus apicis is fractured in a saddle nose it is best to place a cartilage

transplant over the crus apicis, and ivory or cartilage over the saddle. Cartilage and ivory transplant should rest over the periosteum.

Operation for Raising and Narrowing the Nasal Bridge in Saddle Nose: After separating the periosteum over the nasal bones, use a bayonet saw and work from before backward and as near as possible to the inner articulations of the nasal bones. Then with a large double-edged knife, curved in its long axis, an incision is made under the ala at the lower and outer angle of the pyriform opening of the bony nose, thus elevating the skin and periosteum of the nasal process of the superior maxillary bone as far as the root of the nose. Then with an angular saw, cut through the bone of the nasal process of the superior maxilla in the nasobuccal line (sculptural line) running from the margins of the lower and inferior angle of the pyriform opening diagonally upwards to its articulation with the frontal bone and about 5 m.m. or a quarter-of-an-inch away from the inner margin of the orbit, at the same time keeping the left index finger over the inner angle of the eye, protecting it and the lacrimal sac from injury. Then saw through the bone on either side until the unanesthetized mucous membrane is reached (the patient will experience pain, which is the signal to stop) and by pressing with both thumbs towards the middle line against the divided processes, the fracture of the lateral walls is accomplished, and at the same time the nasal bones and frontal processes are disengaged from their frontonasal and maxillary attachments, thereby bringing both sides of the nose together in the middle line. This procedure raises, thereby, the bridge of the nose.

The operation for raising and narrowing the nasal bridge in extreme saddle noses is not only disappointing, but involves much needless surgery, not commensurate with the benefit obtained. Eventually, one must resort exclusively to transplant.

2021 Grand Concourse.

TONSILLECTOMY AND UPPER RESPIRATORY DISEASE (COMMON COLD) IN ADULTS.*

DR. WILLIAM M. GAFAFER, Baltimore.

"Die Tonsillektomie soll nur auf Grund wohlerwogener Indikationen ausgeführt werden und ist dann ein überaus segensreicher Eingriff, der durch keine andere Behandlung auch nur entfernt ersetzt werden kann." P. 244.[1]

Introduction: The question of whether or not an association exists between the presence of tonsillar tissue and the common cold is not infrequently raised. In fact, the otolaryngologist considers frequent colds in an individual one of the possible indications for tonsillectomy. References in the literature to decreases in the frequency or severity of upper respiratory symptoms following tonsillectomy in children are many. The literature dealing with adults, on the other hand, is comparatively scarce. Thus in the case of adults, Smiley,[2] in 1924, reported the information entered by two groups of college students on questionnaires given them. Forty per cent of the group having none or only one cold per year had nose and throat operations, while 54 per cent of the group having four or more colds per year had similar operations. In 1928 Collins,[3] of the United States Public Health Service, published his epidemiological and statistical study of tonsillitis and related throat conditions. He included the results of a study of colds among families of medical officers of the Army, Navy and Public Health Service. The individuals were classified according to whether the tonsils were present or absent. In the adults he found that there was little difference in the incidence of colds in the two groups. Forsythe[4] in the same year recorded the results of a year's observation of about 1,500 male college students, 1,000 with tonsils present and 500 with tonsils absent, paying attention, among other things, to the presence of acute respiratory infections. Of the tonsil-present group, 45 per cent were observed with acute respiratory infections during the year while 62 per cent of the tonsil-absent group suffered similar complaints. Of the tonsil-present group, 68 per cent gave a history of acute respiratory infections (selected) at college entrance while 77 per cent of the tonsil-absent group gave similar histories at the same time. And finally, in 1931, Cunningham[5] compared the histories, recorded between 1920 and 1929, of 12,530 white

*From the John J. Abel Fund for Research on the Common Cold, and the Department of Biostatistics (Paper No. 165), School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Md. The throat examinations were performed by Dr. J. J. Chisolm and Dr. S. W. Egerton, Associate and Instructor in Clinical Laryngology, respectively.

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women age 15 to 34 years, classified according to the condition of the tonsils. There were approximately 8,000 individuals from 15 to 19 years of age, and 3,000 from 20 to 24. One of the questions that each had been asked to answer was if she were subject to colds. The results in this connection may be tabulated as shown in Table I. Whether or not the material shows any association between tonsillar tissue and history of chronic colds may be quickly and easily answered by converting the table into one of the fourfold type as shown in Table II and applying a probability test. Such a test[6] indicates that the material exhibits no association; that is, the tonsil-present group and the tonsil-removed group present no significant difference with respect to history of chronic colds.

TABLE I. INDIVIDUALS WITH HISTORY OF CHRONIC COLDS CLASSIFIED ACCORDING TO CONDITION OF TONSILS (CUNNINGHAM'S DATA).

Condition of Tonsils	Individuals with History of Chronic Colds		Total Individuals
	Number	Per Cent	
Total	719	5.7	12533
Normal	219	5.2	4185
Absent	293	7.1	4134
Pathologic	133	4.7	2836
Remnants	13	4.0	329
Buried or Projecting	61	5.8	1049

TABLE II. TONSILS AND HISTORY OF CHRONIC COLDS (FROM TABLE I).

Tonsils	Individuals with History of Chronic Colds		Total
	Yes	No	
Present	413	7654	8067
Absent	293	3841	4134
Total	706	11495	12201*

*The 329 individuals with tonsillar remnants are excluded.

Present Study: It is the object of the present paper to determine the presence or absence of association between tonsillar tissue and, *1.* frequency, *2.* severity, and *3.* type of attack of upper respiratory disease (common cold) in a group of adults. The basic material is from the John J. Abel Fund for Research on the Common Cold and consists of a part of the enrollment records and a part of the upper respiratory disease symptomatology records of 275 of the individuals that expressed willingness to co-operate with the organization in the common cold research. The individuals of this adult group were students of the School of Medicine, or students or staff members of the School of Hygiene and Public Health. The period of observation is thirty-five weeks long, beginning Sept. 29, 1929, and ending May 31, 1930. For methods and other details that will not be repeated here the reader is referred to the publications of the organization, and attention is particularly directed to Doull, *et al.*,[7] Gafa-

fer[8, 9] and Palmer.[10] The age distribution of the 275 adults with *a.* tonsils present, and *b.* tonsils completely removed, together with information concerning the age at which tonsillectomy was performed, is given in Table III. The age range has a lower limit of 17 years and an upper limit of 59 years.

TABLE III.

Age distribution of 275 adult males and females with *A.* tonsils present, and *B.* tonsils completely removed at *a.* 12 years of age or before, and *b.* after 12 years of age.

Age Group Years	Tonsils Present*†			Tonsils Completely Removed**						
				Male			Female			Total
	Male	Female	Total	At 12 yrs. or before	After 12 yrs.	Total	At 12 yrs. or before	After 12 yrs.	Total	
Total	111	27	138	33	67	105a,b	10	21	32c	137
15-19	3	0	3	1	1	2	1	0	1	3
20-29	92	13	105	31	58	93a	9	16	25	118
30-39	12	10	22	1	5	6	0	4	5c	11
40-49	4	3	7	0	3	4b	0	1	1	5
50-59	0	1	1	0	0	0	0	0	0	0

* Individuals with tonsillar remnants excluded.
† Adenoids out, 7; in, 123; unknown, 8.
** Adenoids out, 56; in, 78; unknown, 3.
a. Age for tonsillectomy unknown: 4
b. Age for tonsillectomy unknown: 1.
c. Age for tonsillectomy unknown: 1.

Frequency of Attack: In the period of thirty-five weeks the 138 adults with tonsils present reported 324 attacks while the 137 adults with tonsils completely removed reported 377 attacks. One individual with tonsils completely removed reported the largest number of attacks, namely, nine. Of the individuals with tonsils present, 74 reported less than three attacks and 26 reported more than three; of the individuals with tonsils completely removed, 62 reported less than three attacks and 40 reported more than three. An application of the theory of probability to these data leads to a result[11] that indicates that there is no significant difference between the tonsil-present group and the tonsil-removed group with respect to frequency of attack of upper respiratory disease. Table IV gives the distribution of the number of attacks of the two groups.

TABLE IV.

Distribution of number of attacks of upper respiratory disease (common cold) reported by 275 adults with *A.* tonsils present, and *B.* tonsils completely removed.

Number of Attacks	Adults with Tonsils Present*	Adults with Tonsils Completely Removed
0	18	10
1	23	20
2	33	32
3	38	35
4	16	27
5	5	6
6	4	2
7	0	3
8	1	1
9	0	1
Total	138	137

* Individuals with tonsillar remnants excluded.

Severity of Attack: Four indices of severity were chosen: in bed, general aching, fever and duration of ten or more days. Table V shows the frequency (per cent) of attacks, classified according to these indices, reported by the tonsil-present group and by the tonsil-removed group. The two groups present no significant difference with respect to the indices of severity.

TABLE V.

Frequency (per cent) of attacks of upper respiratory disease (common cold), classified according to certain indices of severity, reported by 275 adults with *A.* tonsils present, and *B.* tonsils completely removed.

Index of Severity	Tonsils Present*	Tonsils Completely Removed
Total Attacks	324	377
In bed	12.3	14.3
General aching	15.4	14.1
Fever** { first 48 hours†	14.8	13.5
Fever** { later	7.4	6.6
Duration 10+ days	56.5	61.5

* Individuals with tonsillar remnants excluded.
** Includes cases stating fever whether or not a thermometer was used.
† That is, on the first or second day of illness, or both.

Type of Attack: Table VI gives the frequency (per cent) of attacks, classified according to type, reported by the tonsil-present group and by the tonsil-removed group. The two groups show no significant difference with respect to the type of attack.

TABLE VI.

Frequency (per cent) of attacks of upper respiratory disease (common cold), classified according to type, reported by 275 adults with *A.* tonsils present, and *B.* tonsils completely removed.

Type* of Attack	Tonsils Present**	Tonsils Completely Removed
Total Attacks	324	377
Cor+ C— ST—	17.3	17.5
Cor+ C+ ST—	15.4	11.1
Cor+ C— ST+	25.9	23.1
Cor+ C+ ST+	37.3	43.0
Cor— C+ ST+	0.6	2.1
Cor— C+ ST—	0.6	0.5
Cor— C— ST+	2.8	2.7
Presence of:		
Coryza	96.0	94.7
Cough	54.0	56.8
Sore Throat	66.7	70.8

*Cor = Coryza (sneezing, nasal discharge, nasal obstruction, and/or eyes inflamed or watery),
C = Cough
ST = Sore Throat (pharynx, nasopharynx, tonsils, and/or trachea),
+ = present,
— = absent.
** Individuals with tonsillar remnants excluded.

SUMMARY.

1. A group of 275 adults was observed from Sept. 29, 1929, to May 31, 1930, when it was endeavored to secure reports of all attacks of upper respiratory disease (common cold).

2. Of these adults, 138 had their tonsils present, and 137 had their tonsils completely removed.

3. The tonsil-present group and the tonsil-removed group presented no significant difference with respect to *a.* frequency, *b.* severity, or *c.* type of attack of upper respiratory disease (common cold).

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11. The *chi-square* is 4.009 which is equivalent to a probability of .045.

615 North Wolfe Street.

AN UNUSUAL PAPILLOMA OF THE LARYNX.

Dr. Raymond T. Smith, Natchez, Miss.

There has been more literature published in the last decade on papilloma of the larynx, its causes, prognosis and treatment than most any other laryngological subject. This case is cited merely on account of the history and the errors in diagnosis due to the size of the tumor.

The patient presented himself for examination as to the cause of his hoarseness and dyspnea, since on the day before at church he was seized with an attack in which he completely lost his breath and was revived with difficulty, after hypodermic injections of morphin and adrenalin had been given. His history up to that time was as follows:

Eight years ago while watching a gambling game he suddenly became hoarse and lost his voice. The hoarseness continued practically constantly until the attack just mentioned. He consulted different doctors, from whom he got the diagnosis of lues, paralysis of the vocal cords, bronchitis, etc. None, however, gave him any relief. The attack caused him to become more worried and he consulted me, at which time he gave me the following history in addition to the above:

Present Condition: Hoarseness for eight years, difficulty in breathing practically all the time, which at present was so pronounced that he could not sleep, walk, or exert himself at all without completely losing his breath. He was unable to carry on his usual duties as cigar clerk. He has lost about twenty pounds in weight during the past two years. He considered the case entirely hopeless and thought if he could get some temporary relief he would be satisfied. *Past Illnesses:* Usual diseases of childhood. Influenza, four years ago. *Family History:* Father and mother both dead, causes unknown; three brothers living, no sisters. *Physical Examination:* C. W. Reveals an adult male, age 49 years; extremely dyspneic, cachetic, features drawn and anxious facies. Examination of the ears, nose and sinuses essentially negative, teeth extracted; total loss of vision in one eye due to accident many years ago.

Examination of larynx with mirror revealed limitation of movement of the vocal cords, which, however, were normal in appearance.

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Between the vocal cords was a mass which appeared to be papillomatous in character. There was no evidence of cervical or other adenopathy.

The patient at this time was advised to be admitted to the hospital for further examination. This he did not do until March 31. On admittance to the hospital the physical examination was completed. Lungs: Normal on auscultation except for increased harshness in inspiration and expiration. Heart: Normal rate and rhythm, no murmurs. Abdomen: Flat. No palpable tenderness or masses. Reflexes: Normal throughout. Skin: No rashes. Blood pressure: 120/70.

Laboratory Examination—Urinalysis: Albumin, negative; sugar, negative; reaction, alkaline; specific gravity, 1,018; acetone, negative; W. B. C., 8,500; malaria, negative; Wassermann, negative; indican, negative; blood, negative; microscopic, several large clumps of pus. *Differential:* Polys., 72 per cent; small lymph., 24 per cent; large lymph., 2 per cent; transitional, 2 per cent.

Course in Hospital: March 31: Under local anesthesia with the use of the directoscope a small section of the tumor was removed for diagnosis. The laboratory reported benign papilloma.

April 2: Under sodium amytal and local applications to the pharynx an attempt was made to remove the tumor through the oral passage. Before this could be attempted there was a complete respiratory block, causing such extreme cyanosis that it was necessary to perform an emergency tracheotomy. The patient was greatly relieved following this opening and spent a very comfortable night.

April 4: Under local anesthesia the skin and subcutaneous tissues were deadened and a laryngofissure was performed for removal of the growth. On opening the laryngeal cartilage the tumor was seen to have completely filled the glottis and subglottic area. It was macroscopically a pedunculated papilloma attached to the vocal cords in the subglottic area. Complete removal was accomplished at this time and the roots of the tumor were fulgurated with diathermy. The larynx was closed with catgut sutures and no drainage was placed in the wound. Patient made an uneventful recovery.

Results of the operation were a relief of the dyspnea, a louder but slightly coarser voice and an improvement in his general condition.

There was no recurrence of this tumor until Feb. 8, 1931. This was evidenced by slight asthmatic breathing, slight cough. Under local anesthesia an examination of the larynx showed a growth on the margin of the vocal cords. Some of the papillomatous tissue was

removed and patient was much relieved. This operation was performed at the following times: March 15 and June 1, 1931.

On Aug. 9, 1931, the appearance of the growth changed and a specimen was sent to the laboratory for examination. The pathologist's report on this was as follows: "While the greater part of this growth is papillary I find several areas of atypical epithelial cell infiltration in the depths of the tissue submitted. I feel that this should be regarded as carcinoma (squamous cell type)."

On Aug. 19 he consulted another laryngologist, who also took a specimen. The laboratory report at this time confirmed the first report.

Total laryngectomy was done on this patient, Oct. 4, 1931, with complete recovery, with no signs of recurrence up to the writing of this article. Diagnosis: Papillary squamous cell carcinoma.

CONCLUSIONS.

1. That too many of these cases fall into the hands of general practitioners, who make no laryngeal examination.

2. That apparent fixation of the vocal cords in respiration does not mean malignancy without the clinical and laboratory findings.

3. That after clean and total removal that there are usually many recurrences.

4. That these tumors are prone to become malignant.

5. That regular examinations of the tissue removed should be made for malignancy and at the first indication total laryngectomy performed.

Chamberlain-Rice Hospital.

A NEW APPARATUS FOR TREATING SINGERS AND PUBLIC SPEAKERS.

DR. BEN L. BRYANT, Cincinnati.

Phonasthenia in singers and public speakers has long been a problem in the practice of otolaryngology, and more strikingly, perhaps, than in some other phases of our practice, the results of the treatment of this condition seem to vary directly with the amount of specialized training which the otolaryngologist has had in defects of the singing and speaking voice.

We have, therefore, for some time been impressed with the need for a standardized method of treatment embodying the elements long recognized as beneficial in these cases, and which will make possible the successful treatment of these patients, even when the otolaryngologist in charge has not had the opportunity of a prolonged residence in a clinic devoted to singers and speakers. In addition to the obvious advantages of such an arrangement in the daily practice, the benefit to an opera singer or concert singer on tour is apparent.

Perhaps the two most beneficial measures in treating phonasthenia are Faradic current stimulation to the vocal cords and pneumomassage. The physiological basis for this is the same as for other muscles which have undergone trauma and edema, and whose functioning after the insult fails to return to the normal. Just when, where and exactly how to use Faradic stimulation by means of a hand electrode and hand operated switch is a matter requiring considerable special training. With this in mind, we have devised the apparatus known as the Electro Pneumo-Therapy Apparatus, shown in the accompanying illustration.

As will be seen from the illustration, the apparatus consists of a motor-driven shaft carrying a disk containing eight contact plates, equally spaced, from which the Faradic current is delivered. On this same shaft is mounted a large aluminum disk containing eight circular openings, equally spaced and parallel with the contact plates above mentioned. This aluminum disk revolving before a second stationary aluminum disk of the same character creates a siren, the note produced being dependent, of course, on the speed of revolution. The second stationary disk is interchangeable with another containing

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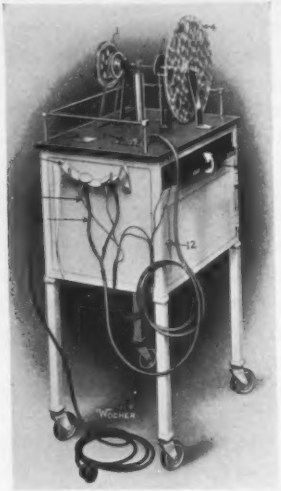
sixteen holes, so as to provide a higher range. From the above mentioned contact plates the Faradic current is delivered to two electrodes mounted on an adjustable neckband to be placed at the sides of the thyroid cartilage. The Faradic current is supplied from a coil, which is mounted, as is the motor which drives the shaft, on the inside of the cabinet below. In the electrode is incorporated a vacuum cup to which air is delivered in a positive and negative



1. Balance wheel. 2. Air pump. 3. Disk with contact plates. 4. Aluminum siren disks. 5. Driving belt from motor. 6. Motor switch. 7. Faradic current switch. 8. Neck band carrying electrodes and vacuum cups. 9. Faradic current lead. 10. Air hose from pump. 11. Rheostat regulating motor speed. 12. Door to cabinet enclosing the motor and Faradic current apparatus.

The following improvements have been made which are not shown in the above illustration:

All movable parts are mounted in closed, ball-bearing housings.

There is a one-quarter-inch mesh wire guard mounted in front of the revolving disk.

The rheostat (11) has been replaced by a grid-type rheostat mounted inside the cabinet and controlled by a small revolving knob, which is the only part of the rheostat control that is visible.

phase, synchronized with the Faradic stimulation. Thus there are two possibilities of operation: If a trained singer is being treated, the motor is set at a speed which will produce from the siren a suitable note. The patient hums this same note and the electrodes having been adjusted, the Faradic stimulation and the pneumomassage may be carried out for any desired length of time with full assurance that the proper phase in the physiological vibration of the vocal cords

will be stimulated. If, on the other hand, an untrained singer or student is being treated, the same result may be achieved by having the patient sing a certain note and matching this note with the siren by means of the rheostat adjustment regulating the speed of the motor. This can, of course, be varied from time to time during the treatment so as to cover any notes with which the patient has difficulty. This means of treatment is offered as a substitute for the former haphazard method of stimulation by means of a hand electrode.

Treatment with the electro pneumo-therapy apparatus is particularly effective in cases of phonasthenia in professional singers, voice students, lecturers, actors, ministers, teachers, public speakers, sales women, etc., and the results in these cases are very striking.

It is noteworthy that practically every patient arises from the treatment chair commenting on the comfortable sensation imparted to the larynx and throat by the treatment, and patients whose chief complaint has been a persistent detonation often find this condition entirely corrected by five or ten minutes of stimulation by the apparatus.

In the subacute laryngitis cases where the vocal cords remain mildly injected, the pneumomassage can be given alone, and it will be noticed that after five minutes of such treatment there is a definite area of erythema surrounding the site of application of each vacuum cup.

We have also noted with a great deal of interest that persistent parasthesias and foreign body sensations often disappear after the use of the apparatus.

This apparatus is made according to my design by the Max Wocher & Son Co., Makers of Scientific Apparatus and Surgical Instruments, Cincinnati, Ohio.

Doctors Building.

THE USE OF DISULPHAMIN IN CERTAIN OTOLARYNGOLOGIC CONDITIONS.

Dr. Farel Jouard, New York.

Having witnessed a large number of striking therapeutic results where disulphamin was administered in cases of pneumonia, bronchitis and influenza, it occurred to the writer to use this drug in the treatment of ear, nose and throat infections. Accordingly, some fifty patients suffering with acute and chronic tonsillitis, acute and chronic sinusitis, acute and chronic otitis media, acute and chronic laryngitis, parotitis, diphtheria and Vincent's angina, respectively, were given disulphamin in one-gram doses, dissolved in water, every two hours for one, two and three days, as the exigencies of the case demanded. In children the dose was proportionately reduced, and never were more than six doses given within twenty-four hours.

The results, which have been summarized in this brief paper, are interesting, in that they not only throw light on the action of the drug, but suggest a new and, to a certain extent, abortive treatment of several serious affections of the ear, nose and throat.

Of the chronic conditions mentioned it may be said that little, if any, improvement was noted from the dosage and manner of administration. In the acute manifestations, however, where fever was present, and especially where the fever was high, say 102° F. or above, the effect was prompt and decisive. The fever, headache, pain and malaise usually began to subside after the second or third dose, sometimes later. Where there was no fever, the results were, on the whole, less gratifying, although occasionally the patient felt decided benefit.

In acute otitis media, the cases selected had fever, pain and inflamed tympanic membranes but no bulging as yet. The majority of these cleared up quickly. A few went on to suppuration and required paracentesis.

In the sinus cases, the frontal sinuses were mostly involved; several showed a pansinusitis. These all cleared up without developing empyema.

The cases of acute tonsillitis, laryngitis and one of parotitis were accompanied by influenza symptoms. These showed prompt relief with administration of the drug. There were three cases of diph-

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theria treated which received antitoxin in addition to disulphamin. However, they were given disulphamin first and showed marked improvement from this medication; it was not until the cultures returned positive that antitoxin was given as a precautionary measure. The one case of Vincent's on record was also treated successfully with disulphamin until the organisms were identified, then neoarsphenamin was administered, also as a precaution. In all the throat cases mentioned the patient was instructed to gargle with the solution before swallowing it.

From the above it would appear that the drug is most potent at the onset and during the early stages of the disease, when communication between the diseased organ and the rest of the system is free and open, when the general reaction and the local one go hand in hand. Later, when lymphatic drainage becomes blocked, or slowed down, it is more difficult to affect the diseased area by general medication. This is especially so when suppuration sets in, or the infection becomes chronic; a partial walling off taking place in either case.

The writer is convinced that a great majority of infections can be aborted with this drug if treated early enough. The above theory of focal-systemic communication and isolation may not be capable of scientific demonstration, but it affords a practical basis for the avoidance of much serious illness. The only drawback, if it can be called such, is that the physician never knows how unfortunate an outcome his patient was headed for, and that the latter often recovers before the physician can even establish a diagnosis.

1 West 89th Street.

International Digest of Current Otolaryngology.

Editor:
DR. MAXWELL FINEBERG, St. Louis.
Collaborators:
Prof. G. Bilancioni, Rome.
Mr. W. S. Daggett, London.
Priv. Doz. G. Kelemen, Budapest.
Dr. H. C. Rosenberger, Cleveland.

Dr. A. Fumagello, of Madrid, Spain, Secretary-General of the International Oto-Rhino-Laryngological Congress which is to meet at Madrid, Sept. 27, 1932, kindly requests that the American colleagues who expect to attend the Congress promptly forward their notification forms in order to avoid delay and difficulties at the last moment.

Sawatari, in the *Zeit. f. H., N. u. O.*, 30:228, 1931, writes on The Influence of Diet on Tonsils. Tonsils of rabbits have been affected either progressively or regressively by means of the nutritional intake; however, this is seen not only in the tonsils but in all of the lympathic tissue. In the tonsils one sees an inflammatory reaction, hardening of the vessels, infiltration of pseudo-eosinophiles in the follicles which wander into the crypts. The theories up to the present do not sufficiently explain these phenomena. One may say that the tonsils in a broad way have something to do as a protective mechanism in digestion. KELEMEN.

Abramow, in the *Zeit. f. H., N. u. O.*, 30:197, 1931, discusses the effects of tracheobronchoscopy on respiration and blood pressure. Tracheobronchoscopy, whether used for therapeutic or diagnostic purposes, is an important procedure, in that it does influence the respiratory and circulatory systems. The variations in the blood pressure and the changes of rhythm in respiration are produced by reflex stimulation of the mucous membrane of the air passages. The cutting through of the superior laryngeal nerve and the vagus nerve causes the disappearance of these reflexes. The shortening of the time of bronchoscopy seems to make no difference to the blood pressure and respiration. Cocainization of the mucous membrane seems to minimize temporarily the reflexes on the heart and breathing apparatus and eases the passing of the tube. For this reason cocainization must be strongly recommended. KELEMEN.

Tobeck, in the *Zeit. f. H., N. u. O.*, 30:182, 1931, presents an article on the histological reconstruction of lymph epithelium following X-ray exposure. The so-called lymph epithelium, when exposed to X-rays rapidly undergoes macroscopic diminution in size; microscopically there is a rapid breaking down of the epithelial elements, while the lymphocytic portions are rather well preserved. The debris of the broken down cells calls forth a production of granulation tissue in which there is an abundance of plasma cells. This brings to mind the interesting question as to whether the X-ray exposures are therapeutically of any value, and it seems to be the author's opinion that the susceptible epithelial tissue, having been broken down, and scar formed, makes for a less malignant picture than the histological structure of the epithelial elements in themselves would suggest. He also points out that the lymphocytic infiltration perhaps is of some use in breaking down and metabolizing the destructive products of the epithelial elements. KELEMEN.

Waldapfel, in the *Zeit. f. H., N. u. O.*, 30:245, 1931, presents an article on The Pathological and Anatomical Predispositions Which Must Occur Before the Development of a Traumatic Angina. While the bacterial flora is minimal in a normal nose, the insertion of a tampon readily allows every type of organism to be found. At this point it is important to clearly state that the hemolytic streptococcus in pure culture may be found in the previously tamponed or cauterized nose which later develops a traumatic angina. Gauze strips or cotton packs produce an excessive immigration of leukocytes with an otherwise normal epithelium. Wide strips of gauze produce destruction of the uppermost layer of the epithelium with hyperemia and leukocytic infiltration of the adenoids and surrounding glands. If iodoform gauze is used, one finds the same results; only sometimes the changes are more marked, resulting in small circumspect areas of necrosis. The mucous membranes, following tamponade, in cases which later developed angina, were found to be disturbed to a high degree and small areas of necrosis in the mucous membranes were apparent. Some little distance away, the glands and lymph channels are found with unmistakable thrombus formation. In this changed tissue, it is possible in almost every case, to find a clear culture of hemolytic streptococcus. While the parenchyma of normal tonsils never contains bacteria, the same tonsil following nasal tamponade, even when it clinically produces no symptoms, contains streptococci in the follicles. KELEMEN.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Dec. 1, 1931.

Catheterization of the Natural Ostium of the Maxillary Sinus Versus Inferior Meatal Puncture. Dr. Henry Dintenfass.

The five ways of entering the antrum for its investigation are, first, through the alveolar margin after a tooth has been extracted; second, puncture through the anterior wall in the canine fossa; third, puncture through the middle meatal wall; fourth, puncture of the inferior meatal wall; and fifth, through its natural ostium.

The alveolar margin route and the canine fossa route were employed years ago but later became obsolete. Puncture of the middle meatal wall, while often successfully accomplished, carries with it the danger of entering the orbit and the possibility of hemorrhage from an anomalous vessel and, therefore, cannot be used routinely. This leaves two methods, the inferior meatal puncture and catheterization of the natural ostium for consideration in antral investigation. There has been much controversy as to which of these methods is the most suitable. The purpose of this article is to consider their respective merits.

In considering the arguments in favor of the inferior meatal puncture the simplicity of performance and successful attainment in practically all cases is at once apparent.

The drawbacks to this procedure are the dangers of air embolism, hemorrhage, shock, pain, emphysema and infiltration of the soft parts and obstruction of the needle.

In considering the advantages of ostial catheterization over the meatal puncture it is readily seen that the above dangers are completely eliminated. Sliding the cannula through an opening instead of the necessity of piercing tissue, bony or otherwise, does away with these objections. However, the practicability of using the natural ostia for irrigation also presents certain definite limitations. The presence of a markedly deflected septum, a low position of the middle turbinate obscuring the hiatus, the bulla and the uncinate process, a deep infundibulum or an unusually large bulla will prevent the proper insertion of the cannula. Then, too, because of the close relationship of the orbit to the middle meatus the improper introduction of the instrument may result in forcing it into the orbital cavity. A great many times the ostium of the maxillary sinus it not really an opening like a doorway but rather takes the shape of a canal, passage or tube pointing laterally, downward and in a forward direction, increasing the difficulties of entering the sinus through the natural orifice. For that reason Hajek advises the preliminary introduction of a flexible silver probe to ascertain the shape of the anatomical parts concerned before the cannula is inserted. In this connection the presence of accessory ostia should also be mentioned. When they exist, which happens in about 18 per cent of the cases, they afford a more direct communication between the maxillary sinus and the middle nasal meatus and can be used to catheterize the sinus in exploration of that cavity.

In summing up whether inferior meatal puncture or ostial catheterization is the more practical method it should be remembered that ostial catheterization obviates the dangers and drawbacks of inferior meatal puncture but cannot be used as frequnently because the anatomical configuration will not always permit. It also demands a more thorough knowledge of the anatomy of the lateral nasal wall together with some degree of skill and experience. Nevertheless, when it can be accomplished it is performed with such apparent ease as to evoke favorable comment on the part of the patient. Therefore, in the diagnosis of diseases of the maxillary antrum, ostial catheterization should always be attempted before the inferior meatal puncture is resorted to.

DISCUSSION.

DR. LAESSLE: Dr. Dintenfass' paper and pictures were interesting and instructive. But to call attention to his method of approach to the natural ostium of the maxillary sinus, I feel that this is not the choice method of entering the antrum, due to the fact that we are prone to make many false openings. I have found, even after the infraction of the middle turbinate, I was only able to enter what I felt was the natural ostium in only 5 to 10 per cent of the cases.

DR. COATES: I congratulate Dr. Dintenfass on his graphic presentation of the different methods of entering the maxillary sinus for purposes of diagnosis and irrigation. Undoubtedly, in certain cases, the method of irrigating the maxillary sinus through the natural ostium has advantages. It should be, if properly done, less subject to danger of death from air embolism than when puncture of the inferior meatus is made with a small needle. When there is a large accessory ostium, entrance through the middle meatus is easy, but I still have doubts that a very large percentage of irrigation can be carried out through the normal ostium. As Myerson has well shown, it depends largely on the size, type and position of such an ostium whether it can easily be catheterized or can be catheterized at all. Many of my patients object to this type of irrigation and prefer the needle puncture in the inferior meatus. Cases needing maxillary irrigation quite frequently have the middle meatus occluded by a septal deviation, middle turbinate hypertrophy or hyperplasis or polypi. These are the cases where it is most difficult or impossible to use this route. I feel convinced also that in some of the cases where it is claimed that the natural ostium has been catheterized, in reality entrance has been made forcibly through the pars membranacea.

DR. WAGERS: I have greatly appreciated Dr. Dintenfass' presentation of this subject. The practical purpose of these various methods of entering the antrum is to enable one to determine whether or not infection is present.

To accomplish the same result I always shrink the mucous membrane of the middle meatus and apply suction. If this fails to bring forth purulent exudate and I have any reason to believe that such an exudate may be present, I then make the inferior meatal puncture and irrigate.

I have catheterized the natural ostium in some instances, but I have not found this method of entry possible in many cases and I have never had any unpleasant results from the usual puncture and irrigation.

DR. DINTENFASS (in closing): I wish to express my appreciation to those who took part in the discussion. I am very sorry that Dr. Coates did not hear the beginning of my paper, in which I mentioned the presence of accessory ostia.

In children the natural ostia are relatively much larger than in adults. In adults, although the natural ostia are very difficult to enter, accessory ostia are found in about 18 per cent of cases and can be utilized in exploration of the antrum. Therefore, opportunity for catheterization is afforded whether it be through the natural or accessory ostium.

Dr. M. C. Myerson mentions the three differently placed positions of the ostia: the vertical, oblique and horizontal. The vertical placed ostia are quite accessible and can be sounded by the single curved instrument of the Siebenmann type, whereas the horizontal and the obliquely placed ostia are very difficult to catheterize. In the latter better results are obtained with the Pierce and the Myerson Modification of the Pierce Cannula.

One must always remember that the position of the infundibulum in its entire extent is very close to the orbital cavity, so that the improper use of the cannula in ostial catheterization might easily injure the orbital wall.

Case Report: Von Recklinghausen's Disease of the External Auditory Canal. Dr. Henry Wieder.

(Published in full in this issue.)

DISCUSSION.

DR. GREENBAUM: When Dr. Wieder telephoned me several days ago and asked me to discuss this case, I told him there was not much to say about it, and after hearing his report I have still less to say.

When I saw this case for the first time in June there was something about the ear growth that suggested a further study. One never sees a growth of this nature without finding more on the body, and on further examination that was exactly what I found. Aside from the forms of neurofibromatosis mentioned by Dr. Wieder, one can have a larval form in which no tumors are present. In addition to that you may have mental changes of a retrograde type.

Von Recklinghausen's disease is not considered an endocrine disturbance. The growths are not real fibromata; they are now believed to be neoplastic proliferations of the peripheral neuroglia.

Removal of these tumors is not dangerous since they are not sarcomata; regrowth following partial or complete removal is rare. There is nothing to be done for this condition except mechanical removal of the growths. In this instance the aural growth is in the field of the otologist. It is preferable to leave the lesion in this patient's ear untouched, but if she demands treatment, destruction by electrocoagulation is the preferable method.

DR. STOUT: I am glad Dr. Wieder brought this case here for the benefit of the younger men present. They will probably not see more than one case of this type in their lifetime.

I saw this case in the operating room when Dr. Wieder had it up for examination and possible operation, and I concurred with him that he had better not operate at that time until further studies could be made. For one thing, there was an undeveloped mastoid on the side that had the tumor formation. It is interesting to consider that this tumor might have had something to do with the undevelopment of that bone.

DR. WIEDER (in closing): Just want to say that removal of this tumor is not the easiest thing in the world and I do not know how good results we will get. We are going to let Dr. Greenbaum remove as much as he can by coagulation. If she is satisfied we will let her go home. If not, we will move the ear forward, as in a radical mastoid operation, remove the growth from the canal and then throw the ear back again, but first we will see if she will be satisfied with coagulation.

Case Report: Giant Cell Sarcoma (Giant Cell Tumor) of the Septum. Dr. Henry Wieder.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Physiology of the Antrum. Dr. Robert J. Hunter.

(Published in full in THE LARYNGOSCOPE, *March, 1932.)*

DISCUSSION.

DR. LEWIS: Dr. Hunter's paper shows him to be a man of courage in bringing before a clinical group a problem in academic physics.

Since we are dealing with volume containing cavities and orifices, we may begin to study the presentation by analogy. Let us consider the antrum a container with rigid sides, a box, with a small hole or orifice in one of the sides about half-way from the top to the bottom of the side wall. If the container is filled slowly with fluid, it will be necessary for the fluid level to reach the lower margin of the hole before fluid will run out of the hole. Alteration in pressures on the outside will have no effect on the drainage. Now let us tilt the vessel to its side which has the hole in it. We will have drainage if the hole is large enough to let air into the space above the fluid level, or if we lower the outside pressure.

If we make a tube whose inside diameter changes similarly as two nozzles whose small diameters meet, that is, the diameter changes from large to small, and back to large, as we go from one end to the other. This will be similar to the Venturi tube. If a hole is made in the tube at its small diameter, and this tube is placed so that the hole is opposite the orifice in the container's side, we have a set-up which is analogous to the physiological antrum described by the speaker.

If air is blown through this tube, the pressure in the ostium and the container becomes reduced, and if there is fluid above the ostium, it will flow into the tube until the pressure in the container is equal to that in the tube at the narrow diameter. If the flow of air in the tube is intermittent, the atmospheric

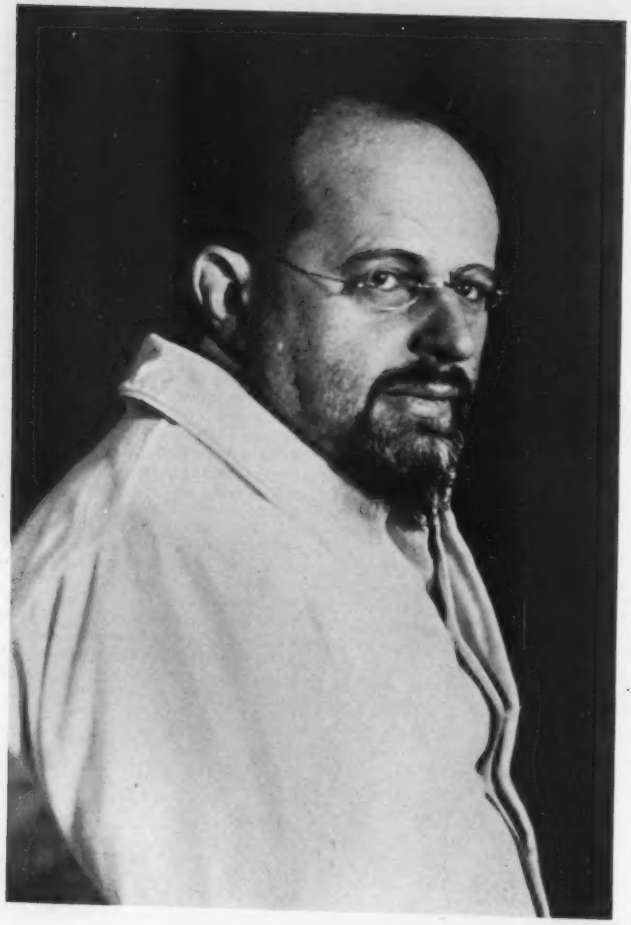
air will equalize the lowered pressure in the container when the flow of air through the tube stops. A repetition of this procedure would in time establish a fluid level at the orifice, but it would not go below this level unless the container were tilted and then the air current could flow through it in opposite directions, and produce a lowered pressure at the orifice in both directions of flow. This would decrease the emptying time.

The antrum is a container which has rigid walls. It has an orifice about half-way up its side wall and, as Dr. Hunter has pointed out, there is an anatomical constricted tube through which air passes on respiration. The antrum may be tilted, and is tilted when the patient bends his head from side to side, and lies down. In addition to the above there is an added mechanism. The cilia of the lining cells sweep the secretion towards the ostium. If the secretion is swept into the ostium and occludes it, a decreased pressure in the tube will pull the secretion out into the nasal airway. If the level of the fluid is above the ostium, the drainage may be accomplished as described previously.

Dr. Hunter demonstrated the effects of moving air and fluids on pressures in constricted tubes. He also pointed out the anatomical analogue of the constricted tube. The investigative work to be carried on will require considerable concentration on methods, apparata and technique. It will necessitate development of methods for measurement of low velocity and pressure heads in moving air currents, along with checks on these measured values.

Dr. Stout: I would like to call attention to the fact that while doing a Caldwell-Luc or Denker operation, if you are careful in removing the bony wall of the antrum and leave the mucous membrane intact, you will notice that with inspiration and expiration there is movement of the mucous membrane, showing that there is positive and negative pressure with every cycle of respiration. I recall some years ago in operating with Dr. John H. Gunter that we particularly noticed a very marked movement of the mucous membrane. Of course, if the bone is in place I do not think the mucous membrane moves, although there is positive and negative pressure in the antrum with the respiratory cycle if the ostium is open.

Dr. Hunter (in closing): Answering Dr. Stout, that same problem has come up in a study of intracranial pressure. As soon as you uncap the antrum in a Caldwell-Luc, you have a new factor involved. It is the same thing in the cranium. The cranium is a closed cavity. When opened we can see with every inspiration a drop of intracranial pressure. That does not prove to me that the same goes on with the cranium closed. Nobody has been able to answer that yet. To do so one should screw a manometer into the skull and seal it in place. When you uncap the lateral wall of the antrum, leaving the mucous membrane intact, you bring in the factor of outer air pressing on it and you also have an elastic wall to consider. I do not believe you can draw any conclusions from Dr. Stout's observation without further study.



GUSTAV ALEXANDER.

GUSTAV ALEXANDER

1873-1932

On April 12, 1932, Prof. Dr. Gustav Alexander, of Vienna, was assassinated on the street between his home and the Policlinic by a former Czechoslovakian patient, Johann Sokoup. Twenty-two years ago a first attempt at assassination by this same man was frustrated. The claim and grudge of this ex-patient is stated to be that he suffered a facial deformity as the result of an operation in 1905, for saddle-nose. He had been remanded and confined to a sanitarium for years following the first attempted assassination.

Through this ruthless murder the otological world has lost its outstanding and most distinguished figure. He was stricken in the period of his finest professional experiences and greatest accomplishments. Endowed with a keen, original and philosophical mind, a prophetic, scientific vision, a rich and versatile cultural background, an unusual histo-pathological, anatomical, neurological and otological training, a remarkable urge for research, a tremendous and indefatigable work-energy, great humane instincts, and a genial yet forceful personality, he has enriched the world of science generally and otology especially with a masterful and enlightening array of contributions second to none in his generation, and has erected by his own manifold labors in otological, anatomical, neurological and sociological fields a permanent monument which must justly claim the everlasting gratitude of the entire human family.

Gustav Alexander was born in Vienna, Dec. 18, 1873, received his doctor's degree in 1898, was appointed demonstrator of anatomy by Zuckerkandl while still an undergraduate in 1895, named assistant in the Alberts Surgical Clinic in 1900, and also began his otological work in the Politzer Clinic in 1900, continuing his service there until the retirement of Professor Politzer. In 1903, he was made Dozent; in 1909, he received the title, "Professor Extraordinarius"; and in 1919, "Professor Ordinarius."

In 1907, he was appointed to succeed Prof. Urbantschitsch as head of the Department of Otology of the Vienna Policlinic, the position which he occupied at the time of his death.

It was largely through his personal efforts, scientific attainments and genius as a lucid and authoritative teacher that the Ear Clinic of the Policlinic of Vienna has grown to recognition as one of the outstanding units in this special field in the world.

Not only his clinical and teaching accomplishments dominated and influenced the progress of otological science, but he was also a prolific contributor to its literature, as evidenced by over two hundred articles, monographs and books.

His field of observation and research was a wide one and included anatomy, embryology and physiology of the hearing organs; pathological anatomy, and clinical otology.

His contributions to the anatomy of the internal ear offered much new data, made possible by his profound knowledge of otological fundamentals, keen perceptions, and mastery of anatomical technique. He devised a new technical method for the study of the descriptive and topographical anatomy of the labyrinth, the preparation of celloidin sections and of wax-plate reconstruction, thereby facilitating detailed research in anatomy of the ear.

Comparative anatomy and embryology of the ear were favorite fields which he has enriched by numerous monographs. He even pursued ethnographic studies and made some interesting observations on the temporal bone of the American Indian and the Negro.

Of his important work in the physiology of the labyrinth may be mentioned his conclusion that the statolith apparatus, in its usual performance, plays no prominent role in space orientation.

Twelve original monographs of equal significance are comprised in his physiological contributions.

Pathological histology of the internal ear, however, was the field to which he was most devoted, as attested by over forty papers and monographs. Here we emphasize the value of his findings in the sacculo-cochlear type of deafness, as he described it in the albino cat, deaf dog, dancing mouse, and also in congenital deafness of the human. He was the first to describe primary atrophy of the neuro-epithelium of the nerve-ganglion apparatus producing profound deafness and in some instances tracing the origin of such atrophy to fetal disease. He also indicated that the pathological changes in otosclerosis, in essence, may be of congenital character; this opinion is gaining in otological consideration today.

As a master of clinical otology, Alexander's opinions and conclusions were always regarded with respect and finality.

He was keenly interested in the problems of congenital deafness and the education of deaf and hard-of-hearing children. It was in this field of special sociology that his humane impulses and kindly co-operation were actively engaged. His observations of the deaf child were made during many years of regular service as school otologist. He organized kindergarten classes for the early training of congenitally deaf children.

For many years he was actively identified with the Vienna School for Deaf Children and regretfully deplored the fact that financial circumstances compelled the disbandment of this school last year.

His book, "Ear Diseases in Children," is a classic; his "Handbuch der Neurologie des Ohres," produced conjointly with Marburg, has stimulated modern otology to an increasing recognition of the value of these associated specialteis; his co-editorship of the *Monatsschrift fur Ohrenheilkunde* has helped to maintain the high standard of this veteran among otological periodicals.

Added to these varied scientific accomplishments, his cultural inclinations and talents were also worthy of special recognition. He was a great lover of music, an accomplished pianist, and with his son and daughter (an expert cellist), often played in trio at his home.

He was an enthusiastic connoisseur and collector of art objects, specializing in paintings, bronzes, old clocks, silversmith creations, and early tiles. His otological library was one of the most complete privately assembled in this field.

He was child-like in his enthusiasm over a new acquisition and was at his best when he could discourse with an appreciative listener about it.

Last Christmas he expressed his joy in an interesting letter to me, with ethnographical comments, on receipt of a small collection of Indian arrowheads and axeheads which I sent him as a small token and holiday greeting.

So, in this man of profound and versatile accomplishments we recognize, next to Politzer, the finest product of the Vienna otological school, and unhesitatingly rank him as the greatest otologist of his day. His enrichment of otology and his influence in its progress will be an everlasting legacy.

To his bereaved family and intimate circle we offer our heartfelt condolence. We have enjoyed the hospitality of his home, have had the privilege of meeting the members of his talented family, and have appreciated the stimulus and charm of this fine influence.

To me, personally, the passing of this remarkable man recalls dear and early memories and associations, for I, too, was an assistant in the Politzer Clinic for a short period, preceding Alexander and Neumann there. I enjoyed the opportunities and privileges of service under that great master and pioneer in otology, Adam Politzer — Politzer and Alexander — "Pax Vobiscum."

MAX A. GOLDSTEIN.

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